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ARCHIVES OF SURGERY

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THE MANAGEMENT OF CRANIOCEREBRAL INJURIES *

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AND

ALBERT S. CRAWFORD, M.D.

DETROIT

During the past decade a number of valuable papers dealing with craniocerebral injuries have appeared. The importance of this problem is being realized more and more in this era of great industrial expansion with the congestion of rapidly growing cities and the constant increase in the speed of travel. Reports of critical analyses of series of cases have helped to make the important features of the diagnosis more clear, and a more effective management of cases of injury to the head has gradually evolved. In spite of this improvement in results, members of the medical profession should strive to do better, and their experience should be made available to others.

We report a series of 441 cases of craniocerebral injuries in which the patients were treated in our hospital during a period of a little over ten years. Only cases of injury to the cranium or cranial contents are included in this series, all questionable cases of concussion and simple injury to the scalp being omitted. Our series probably represents fairly well the experience in an average general hospital in a large, rapidly growing industrial city. The cases come from both industrial and civil life, and a fairly large number have been followed in the outpatient department until the patient has been entirely cured. An effort has been made to keep in touch with the other patients by letter.

An attempt has not been made to record all the collected data, and only those observations are given which may have some practical value in diagnosis and treatment.

GENERAL DATA

For the sake of a background, we have made an attempt to secure some general data regarding injuries to the head in industrial and civil life in the United States. In regard to the incidence of injuries of the head, the available figures are of interest: In 1927, the United States Department of Labor, Bureau of Labor Statistics,[1] reported that in 1917,

* From the Surgical Department of the Henry Ford Hospital.

1. Bull. 425, Bureau of Labor Statistics, U. S. Dept. of Labor, 1927.

there were 1,558,318 industrial accidents reported from forty-three states, of which 11,388 were fatal. Each year the totals fluctuated, being less during 1921 and 1922; then rising slowly till in 1926 the total was 1,698,494 with 10,537 fatalities. In 1926, the New York department of labor [2] reported 76,216 compensable disabilities of which 3,997 were injuries to the head, with 300 deaths (7.5 per cent). Males outnumbered females 14 to 1. These figures doubtless are accurate, for they have been reported by the various state industrial commissions; however, a certain number of accidents are not reported. At the present time, accidents in civil life are not being reported to any one bureau, so that figures for the total number of accidents are not available. As there is no uniformity of the data being given, they are without much real value to us. The National Safety Council [3] is rendering an invaluable service in studying present conditions of accident hazards all over the nation and planning methods of improvement. Although they are not yet able to give total figures with definite accuracy, they have compiled some estimates, based on known reliable data, which are of interest. During 1926, there were about 23,000 deaths from automobile accidents (probably from 600,000 to 700,000 total) in the United States, 24,000 deaths from the industrial accidents (probably a total of about 3,500,000 accidents) and 43,000 fatalities from all other accidents. This makes a total of about 90,000 deaths from accidental causes in 1926.

The National Safety Council reported that at present the death certificates of the health departments, which should be accurate and complete, are lacking in details, and often are almost worthless. The medical profession should take the responsibility of making possible a comprehensive system of reporting all accidents, accurately and uniformly, so that the data can be available by localities or states and according to traumatic agencies and other etiologic factors. From the data available, we can be fairly certain that there is a definitely rising rate of accidents each year and a corresponding increase in deaths from injuries of the head. The percentage of such injuries is roughly from 7 to 20 per cent.

ETIOLOGY

The table on traumatic agents (table 1) shows the distribution of our cases according to the agent, age and sex. The results are similar to those in other series. The automobile was responsible for 54.2 per cent of the injuries, which is considerably higher than in any other series in which the agent was mentioned. This fact may have a significant bearing on traffic problems in our city, and suggests that the point

2. Bull. 146, New York Dept. of Labor, July, 1926.

3. National Safety Council, Dept. of Public Safety Division. Personal communications with C. E. Robb and George Opp, safety engineer.

might well be brought out in future reports from other large hospitals in this and all large cities. The next most frequent cause of injury were falls, 20 per cent, and blows, 18.4 per cent. The ratio of males to females in the whole series was 3 to 1, and the number of males varied in the different groups from 63 per cent injured by automobiles, to 88 per cent by falls and 96 per cent by blows. As to age incidence, persons in the first four decades were most affected. The average age for the series was 30. In 1921, Stewart [4] reported 6,135 cases of injuries of the head in New York City, of which 617 consisted of fractured skulls. In this series, 65 per cent were men, and the average age was 36.

TABLE 1.—*Traumatic Agents (Age and Sex)*

Age (Decades) Years	Automobile		Fall		Blow		Street Car		Train		Gunshot		Foreign Body		Not Classified		Totals		(Both Sexes)
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	
0 to 10	38	17	17	4	1	2	..	..	..	..	..	..	..	..	1	..	57	23	80
11 to 20	19	11	6	1	3	..	..	..	..	..	..	1	..	..	2	..	30	13	43
21 to 30	29	25	23	3	23	1	2	1	..	..	1	..	1	..	5	..	84	30	114
31 to 40	28	10	16	1	29	..	3	2	1	..	..	..	..	..	2	2	79	15	94
41 to 50	20	11	5	..	12	..	1	1	..	..	..	..	..	..	1	..	39	12	51
51 to 60	4	7	8	..	9	..	..	..	..	..	..	..	..	..	1	..	22	7	29
61 to 70	10	7	1	1	..	..	2	2	..	..	..	..	..	..	1	..	14	10	24
Not recorded	3	..	2	..	1	..	..	..	..	..	..	..	..	..	..	..	6	..	6
Totals	151	88	78	10	78	3	8	6	1	..	1	1	1	..	13	2	331	110	
Totals (both sexes)	239		88		81		14		1		2		1		15		441		441
Percentage of 441 cases	54.2		20		18.4		3.2		0.2		0.4		0.2		3.4		75	25	100

TABLE 2.—*Location of Injury*

Vertex							Combined Vertex and Basal
Right Temporal Lobe; Partial	Left Temporal Lobe; Partial	Occiput	Frontal	Both Sides	Not Classified	Basal	
58	50	47	82	7	134	25	38
Total (by location)					378	25	38
Percentage of total series					85.7	5.7	8.6

LOCATION AND TYPE OF INJURY

We have classified the fractures of the skull as basal and vault fractures. The latter type is subdivided into fissured (linear and stellate), comminuted, diastatic, depressed and perforated. The location of injury is shown in table 2: 85.7 per cent were in the vertex; 5.7 per cent basal, and 8.6 per cent combined. Stewart's series showed fractures of the vault in 66 per cent and basal fractures 34 per cent. Besley's [5] series of

4. Stewart, J. W.: Fractures of the Skull, J. A. M. A. **77**:2030 (Dec. 24) 1921.

5. Besley, F. A.: A Contribution to the Subject of Skull Fractures, J. A. M. A. **66**:345 (Jan. 29) 1916.

1,000 cases in Chicago, 1916, showed vault fractures, 33 per cent, basal fractures, 34 per cent and combined fractures, 33 per cent. In 1923, Brown and Strecker [6] reported 100 cases in Philadelphia, of which 49 per cent were fractures of the vault and 30 per cent were basal fractures. The probable explanation for our relatively low percentage of basal fractures is that we have not, as a routine, taken the large number of special roentgen-ray positions to bring out basal fractures used by Stewart,[7] and our rate of postmortem examinations has been small as most of the cases were done by coroners' physicians. The point, however, is more or less an academic one, as the factor of real importance is the damage to the internal structures. The fracture of the skull is secondary except when it is compound, depressed or stellate with driving in of bone fragments, and when it tears across an important blood vessel or into an infected sinus.

We have adhered to the old classification of cerebral injuries: concussion, contusion, laceration and laceration and hemorrhage. This

TABLE 3.—*Clinical Diagnosis*

	Concussion	Contusion	Laceration	Laceration and Hemorrhage	Totals
Without skull fracture	257	7	5	..	269
With skull fracture	94	24	49	5	172
Totals	351	31	54	5	441

method of classification is inexact, being based mostly on the clinical signs and the course of the condition. Classifications based on observations of the spinal fluid, such as Rodman and Neubauer's [8] and Jackson's,[9] are probably more exact as to limits, but we have not used them as yet. Table 3 shows the cases according to our classification. The term "concussion" has been used rather broadly, and perhaps includes some cases in which the condition might have been classed as "contusion." The rather large number of cases from this group which had unfavorable sequelae would tend to support this supposition.

6. Brown, H. P., and Strecker, E. A.: Some Observations in the Treatment of Fractures of the Skull, Ann. Surg. **79**:198, 1924.

7. Stewart, J. W., and Luckett: Skull Fractures, Annals of Roentgenology, New York, Paul B. Hoeber, 1925, vol. 6.

8. Rodman, J. S., and Neubauer, B. B.: A Plan of Management of Cranial Injuries Based on a New Grouping of Such Injuries, Am. Surg. **79**:481 (April) 1924.

9. Jackson, Harry: The Management of Acute Cranial Injuries by the Early Exact Determination of Intracranial Pressure and Its Relief by Lumbar Puncture, Surg. Gynec. & Obst. **34**:494 (June) 1922.

DIAGNOSIS

The diagnosis of the mild and severe cases is fairly simple but there is a certain group in which it is difficult to determine the location and degree of injury in the early stages, and to differentiate the factors of edema, contusion, laceration and hemorrhage. We shall not attempt any comprehensive discussion of the technic of diagnosis, nor a tabulation of the clinical data in our cases, but shall give briefly some of the lessons we have learned and emphasize again some of the aids in diagnosis. We have depended mainly on clinical signs and symptoms, lumbar puncture, roentgen-ray observations and clinical courses.

Early Signs and Symptoms.—The following signs and symptoms have been valuable guides:

1. The degree and duration of the primary unconsciousness has been a fair index to the severity of the injury and the probable course, except in cases of secondary hemorrhage and infection. Usually when the unconsciousness lasted several hours or longer, convalescence was prolonged and followed by one or more unfavorable sequelae. There were some notable exceptions; some patients with the most severe injuries made a rapid and complete recovery, while others with mild cases of concussion were incapacitated more or less permanently.

2. Such symptoms as shock, vomiting, color and mental state are usually of help in making a diagnosis, but they are often unreliable because of the great individual variation in reaction and recovery.

3. The "cracked-pot" percussion note of the skull usually indicates the presence of fracture, but it can be deceptive and should not be relied on too much.

4. The external injury to the scalp usually shows the probable site of deep injury and should warn one against middle meningeal hemorrhage or infection from sinuses. Contra-coup injuries, however, are not infrequent and must always be considered.

5. Blood from the cranial orifices (ears, nose and mouth) mean basal fracture unless it is due to external trauma.

6. Conditions of the eye, such as ecchymosis of the lids and subconjunctival hemorrhage, are more apt to be delayed in onset and persist longer if they are of deep origin than if they are caused by external injury. The pupils are important guides to the severity of the injury and to the side on which the lesion is located. They first contract, then gradually dilate, and finally lose the reactions to light. The disks usually do not show changes in the acute stage of the injury. Holman and Scott [10] have emphasized the localizing value of homolateral dilatation and fixation of the pupil.

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10. Holman, Emil, and Scott, W. J.: Significance of Unilateral Dilation and Fixation of Pupils in Severe Skull Injuries, J. A. M. A. **84**:1329 (May 2) 1925.

7. Edema and ecchymosis of the scalp in such areas as the mastoid (Battle's sign) indicate a fracture underneath, but in other regions this sign is unreliable. Hematomas have often been wrongly diagnosed as depressed fractures.

8. The deep reflexes are usually affected early and help to localize the side on which the lesion is located, particularly if there are corresponding changes in the motor and sensory functions.

9. Changes in the motor and sensory functions and pathologic toe signs, the Babinski and other signs, usually occur later; if they appear early, they signify a serious lesion.

Later Neurologic Signs.—1. Steadily deepening unconsciousness after a "lucid interval" usually means intracranial hemorrhage. Localized laceration with edema can closely simulate the picture but is usually

TABLE 4.—*Roentgen Ray and Clinical Diagnosis*

Roentgen-Ray Examination	Clinically Positive Fractured Skull				Clinically Negative Fractured Skull				Total Cases		
	Roentgen Ray Positive		Roentgen Ray Negative		Roentgen Ray Positive		Roentgen Ray Negative		Roentgen Ray Positive	Roentgen Ray Negative	Percentage of Total Cases
	No.	%	No.	%	No.	%	No.	%			
Made	117	33.1	38	10.7	10	2.8	180	53.4	127	227	80.3
	155				199				354		
Not made or not reported	34				53				87		19.7
Totals	189				252				441		100
Percentage of total	42.8				57.2						100

slower in developing and clears up more promptly, and, unlike hemorrhage, responds more or less favorably to hypertonic treatment.

2. Changes in the eyes are extremely significant and should be observed at frequent intervals in serious cases. In our series, the pupil dilated on the side of lesion, and all patients with dilated or fixed pupils died. Nystagmus is an unfavorable sign. Choked disks are usually a late manifestation, but engorged veins and a haziness of the margins of the disks show the earlier stages of compression.

3. Involvement of the cranial nerve occurs early or late and is permanent or temporary, depending on whether the nerve is severed or merely compressed by accumulated fluids. The sixth nerve is reported as being most frequently involved. In our series, the eighth and seventh nerves were most often affected.

4. Progressive motor or sensory changes are important and indicate partial pressure or complete severance of pathways. The early hypertonus and later irritability give way to spasticity and then to weakness and flaccid paralysis in the terminal stages.

5. The deep reflexes usually change correspondingly and help to localize the lesion. The possibility of pathologic reflexes from spinal syphilis and other conditions must be remembered.

6. The vital signs (temperature, pulse and respirations) and blood pressure are indicators of the condition of the medullary centers. These signs should be recorded frequently in the serious cases. Greenleaf [11] has pointed out the localizing value of unilateral elevation of temperature in the axilla opposite the lesion. At times this might be invaluable.

Fig. 1.—Roentgenogram showing multiple fractures with wide separation and some depression in a policeman, aged 31, who was thrown to the pavement by a runaway horse. The recovery was uneventful with no complications and no sequelae reported.

Roentgen-Ray Examination.—Roentgenograms give definite information about the skull and should be taken in all cases of real injury of the head. It gives a clue as to whether hemorrhage or infection is apt to occur. Table 4 shows that in our series roentgenograms were taken in 80.3 per cent of the cases, and 35.9 per cent of these were positive. In 10.7 per cent of the clinically positive cases the roentgenograms were reported negative, and in 2.8 per cent positive in the absence of positive

11. Greenleaf, Paul: Cranio Cerebral Injuries, Illinois M. J. **47**:46 (Jan.) 1925.

clinical symptoms. A negative roentgenogram is not proof that the skull has not been fractured, because the technic may be faulty or the picture may have been taken at an improper angle. One picture may be sufficient, but several are usually necessary to show the details. Clinical observations are of more importance than roentgenograms, except in depressed fractures. Figures 1, 2 and 3 show serious injury; the patients recovered completely.

Fig. 2.—Roentgenogram showing multiple fractures of the occiput with depression in a janitor, aged 40, who was knocked to the pavement by an automobile. He was unconscious a week, and irrational three weeks. He recovered completely and without sequelae.

Lumbar Puncture.—Lumbar puncture is another fairly accurate diagnostic means and usually indicates the presence of blood, freedom of communication and the intracranial pressure (except when there is a block). It is also a valuable therapeutic agent for relief of pressure and for drainage in infections. The observations on spinal puncture may be unreliable and the drainage dangerous in basal injuries in which there is interference with free communication. We advocate its use only when there is a definite indication, but it should always be done slowly and cautiously. Punctures were made 382 times in 148 cases, or in 33.6

per cent of the total number of cases in our series (table 5). The fluid was bloody in 39.9 per cent of the cases in which puncture was performed and clear in 60.1 per cent. In table 5, the cases are grouped clinically according to the three degrees of severity and also according to the pressures. As would be expected, 83.3 per cent of the cases in which death occurred showed bloody fluid. Stewart [7] reported punctures in 26 per cent of his cases with bloody fluid in 90 per cent. Jackson [9] classified his injuries according to spinal fluid pressure as follows: 14 per cent with pressures over 30 mm. of mercury; 34 per cent, from

Fig. 3.—Roentgenogram showing the depressed fracture of the right parietal area in a boy, 3 years of age, who fell 27 feet. A complete recovery without sequelae is reported.

20 to 30 mm., and 44 per cent, from 10 to 20 mm. In our series, 47.3 per cent had pressures under 10 mm.; 39.8 per cent from 11 to 20 mm.; 10.8 per cent from 21 to 30 mm., and 2 per cent above 30 mm.

TREATMENT

Some of the earliest operations on the brain were doubtless performed to relieve the pressure from hemorrhage. Trephination for this purpose was practiced during the early centuries. Until recently, surgical treatment in most of the serious cases was advocated and practiced, but now there is a definite tendency toward conservatism. There seems to be a general agreement as to nonsurgical treatment in the mild, uncomplicated and moribund cases, but there is still some difference of opinion regarding cases of cerebral compression. Statistics are quoted by

Sharpe,[12] Hendon,[13] Kerr,[14] Mixter [15] and others to prove that surgical decompression lowers the mortality rate. On the other hand, Connors [16] and Munroe [17] and others have quoted figures in favor of the conservative form of treatment. This divergence of opinion would seem to indicate that there is much yet to learn in this regard. The publication of more critical analyses of large series would tend toward the solution of this problem.

We shall give briefly our plan of management, touching on only some of the most important points.

Immediate Care.—1. History of Accident and Injury: We stress the importance of obtaining and recording the data in detail from as reliable sources as possible. In this way we may know whether or not to

TABLE 5.—*Lumbar Punctures (Clinical Groups, Pressures and Results)*

Spinal Fluid	Clinically	Number of Lumbar Punctures Done	Number of Cases Punctured	Average Puncture per Patient	Cases Listed According to Pressures (Mm. of Mercury) 0 to 10	11 to 20	21 to 30	Over 30	Cases Listed According to Results Cured	Improved	Not Improved	Treated	Died	Percentage of Deaths in Cases Punctured
Bloody	Not severe	4	4	1	2	2	..	..	3	1	..	..	....	
	Moderately severe	140	40	3.5	15	18	7	..	23	10	3	4	10	
	Severe	79	15	5.3	..	9	4	2	2	2	..	11	73.5	
	Total cases	223	59						Total deaths				15	
	Percentage		39.9						Percentage of cases punctured				25.4	
Clear	Not severe	49	41	1.2	35	6	..	..	29	10	2	..	....	
	Moderately severe	103	44	2.3	15	24	4	1	24	17	1	2	4.5	
	Severe	7	4	1.7	3	..	1	..	2	1	..	1	25	
	Total cases	159	89						Total deaths				3	
	Percentage		60.1						Percentage of cases punctured				3.4	
Grand totals		382	148	2.6					Percentage of total cases punctured				33.6	25.8

anticipate a depressed, stellate or penetrating fracture. From the medicolegal aspect accurate details are essential. We inquire as to whether there has been a previous fracture or epilepsy and what treatment has been administered before admission.

12. Sharpe, William: Observations in the Diagnosis and Treatment of Brain Injuries in Adults, J. A. M. A. **66**:1536 (May 13) 1916; Diagnosis and Treatment of Brain Injuries, New York, J. B. Lippincott Company, 1920.

13. Hendon, G. A.: Acute Injuries of the Brain, Kentucky M. J. **22**:505 (Nov.) 1924.

14. Kerr, H. H.: Management of Fractures of Base of Skull, California State J. Med. **21**:477 (Nov.) 1923.

15. Mixter, W. J.: Fractures of the Base of the Skull at Massachusetts General Hospital, Boston M. & S. J. **177**:518 (Oct.) 1917.

16. Connors, J. F.: Management of Intracranial Injuries with or without Fracture, **81**:901 (Jan.-June) 1925.

17. Munroe, Donald: Therapeutic Value of Lumbar Puncture in Treatment of Cranial and Intracranial Injury, Boston M. & S. J. **193**:1187 (Dec.) 1925.

2. Immediate Survey of the Case: An immediate survey is made by some one of sufficient experience to size up the case quickly and accurately and to order the appropriate treatment. Patients in a state of shock are not unduly exposed and complete examinations and the taking of roentgenograms are delayed until there is sufficient recovery. When there is bleeding from the ears, speculum examinations are postponed and are performed only under aseptic conditions.

3. Immediate Surgical Treatment: Surgical treatment is usually necessary only in cases of superficial hemorrhage or lacerations of the scalp. If the patient is in a severe state of shock, all extensive procedures are delayed, heat applied and dextrose administered intravenously or subcutaneously. The strictest asepsis is maintained, and after the wound has been thoroughly cleaned, it is probed with sterile instruments or gloves. The closure of a wound can usually be accomplished without drainage unless it is very much contaminated. Moribund patients should be absolutely quiet, heat being applied and stimulants, etc., used.

4. Tetanus Antitoxin: Tetanus antitoxin (1,500 units for adults) is given in all cases in which a real wound may have been contaminated with dirt. The possibility of anaphylaxis must be borne in mind.

Roentgen-Ray Examination.—If the patient's condition warrants it, the next step is to take roentgenograms of the skull. Enough are made to give details of the type and location of the damage. Sometimes one or two are sufficient. Basal fractures require a special technic. The roentgenograms are read at once. If the fracture line crosses the middle meningeal groove, hemorrhage can be expected; if it is depressed or enters an infected sinus, early surgical intervention may be necessary.

Medical Treatment (Conservative).—All patients should be hospitalized for a varying length of time, depending on the severity of the injury. Our outline of procedure is:

1. If the patient is in a state of shock, the head is kept low, heat is applied to the body and a 5 per cent solution of dextrose is injected by rectum (1,000 cc.) or intravenously (500 cc.) if urgent; ice is applied to the head, and caffeine is given when necessary.

2. After recovery from the shock, the head is raised and kept up to combat edema. The pulse rate, respirations and blood pressure in serious cases are taken every fifteen minutes for four hours; then the interval is increased to every four hours as the conditions warrant. The nurses are instructed to take the blood pressures; their results are checked at intervals by the interne.

3. A neurologic examination is made when the patient is admitted to the hospital; in serious cases, the results are checked at frequent intervals in order to note early signs of a rapidly progressive lesion. The nurses are instructed regarding the danger signals and should know the

significance of the various changes that occur in the four stages of medullary compression. The pressure must be relieved before the onset of medullary exhaustion. Danger signs are exhibited when the pulse pressure is as high as the pulse rate, or when there is a steadily rising pulse rate and blood pressure with a deepening unconsciousness or restlessness. Unfavorable signs are dilating and later fixed pupils, diminishing and later absent deep reflexes, spasticity, clonus, high temperature and a progressively deepening coma. We are on the lookout for unilateral changes in reflexes and corresponding motor and sensory changes. In basal fractures, signs of meningeal irritation mean hemorrhage or infection. Neurologic checks are performed when the patient is discharged and each time he returns during the period the case is followed up.

4. In the early stages, we do not use morphine as a sedative or homatropine as a mydriatic for fear of masking important signs. Codein, aspirin or phenobarbital are usually sufficient and not harmful. If the restlessness is extreme, however, and the danger of hemorrhage is past, the sodium salt of phenobarbital can safely be administered hypodermically, from 1 to 3 (0.065 to 0.00195 Gm.) grains, paraldehyde 1 to 2 drachms (3.75 to 7.5 cc.) by mouth or 2 to 4 (7.5 to 15.0 cc.) by rectum, given carefully in water, or sedative packs.

5. Lumbar puncture is one of the most valuable diagnostic and therapeutic means; it is used in all cases in which the diagnosis is uncertain, and therapeutically for cerebral compression. It is dangerous when the patient is in a state of shock and when there is a block at the base and high pressure above. In the latter condition, ventricular puncture is a safer method of drainage. Lumbar puncture is performed slowly, the pulse rate, respirations, color and blood pressure of the patient being noted. The patient is in the lateral prone position, and the pressures are taken with a standard mercury manometer. The amount of fluid removed depends on the conditions. If removal produces unfavorable signs, the pressure is read and only a few drops of fluid are removed to determine the color. We usually remove a sufficient amount to reduce the pressure to half of what it was originally. Therapeutic punctures are repeated as indicated. In our series (table 5), the number of punctures per patient was 2.5; the procedure without doubt saved many. Jackson [9] strongly advocated its use and felt, as we do, that it is a more reliable guide than vital signs and blood pressure. Green [18] and Holbrook [19] also advocated strongly the use of lumbar puncture. Besley,[5] on the other hand, pointed out with good reason that if puncture is performed too frequently, it may cause an excessive secretion of spinal

18. Green, T. M.: Management and Treatment of Brain Injuries, Internat. Clinics **2**:239 (June) 1924.

19. Holbrook, F. C.: The Diagnosis and Management of Head Injuries, J. A. M. A. **83**:489 (Aug. 16) 1924.

fluid. This was possibly so in some of our earlier cases, in which a large number of punctures became necessary in order to control the increasing pressure. The Queckenstedt maneuvers may be dangerous in cases of injury of the head, and are not usually indicated.

6. Another valuable means of nonsurgical reduction of intracranial pressure is the use of hypertonic solutions. In our experience, dextrose and saline have proved the most satisfactory, and dextrose has the advantage in that it is less likely to be injurious, furnishes nourishment as well, and is more prolonged in its effect. We use dextrose alone or combined with 15 per cent saline; 25 per cent (from 100 to 200 cc.) or 50 per cent (from 50 to 100 cc.) is slowly injected intravenously, insulin being added if necessary. If the case is less urgent, magnesium sulphate is given, 2 ounces (59.2 cc.) by mouth or 3 ounces (87 cc.) by rectum. Hyper-

TABLE 6.—*Method of Treatment with Results*

Operation		No Operation			
		Medical Treatment		Not Treated	
Survived	Died	Survived	Died	Survived (Transferred)	Died (At Once)
Early operation... 3	13				
Late operation.... 5	1	363	29	5	23
Total..... 3	14	Total, no operation (medical)..... 392		Total, no operation (transferred) (died) 28	
Operations, total..... 22					
Percentage of patients operated on..... 4.3		Percentage, medical treatment to total.. 88.8		Percentage, no treatment to total.. 6.4	
Mortality rate of patients operated on..... 59.1					
Operative mortality rate of total cases in series..... 3.2		Mortality rate (medical)..... 6.6		Mortality rate (not treated)..... 5.2	
Total number deaths, whole series 65		Total mortality rate of whole series..... 14.7			

tonics do not seem to have any appreciable effect on hemorrhage or laceration, but they clear up the edema temporarily. The treatment may have to be repeated from every six to eight hours. Care must be exercised that dehydration does not result from overdoing the procedure. The intake and output of fluids are carefully charted, and the balance regulated according to the conditions.

Surgical Intervention.—As already stated, there is still a difference of opinion regarding the indications for surgical intervention. Series of cases have been reported in which operations were performed on 70 per cent of the patients; in series recently reported, operations were performed on from 30 to 10 per cent. In our series, craniotomy was employed in only 4.3 per cent of the total number of cases (table 6). Subtemporal decompression was performed for middle meningeal hemorrhage and craniotomy elsewhere for definitely depressed fracture, debridement for compound fractures or penetrating wounds with contamination, drainage of abscess of the brain or a compound fracture into

an infected sinus and for late epilepsy with a localized depression. It does not seem necessary to elaborate on the technic of decompression. Cushing's time honored subtemporal approach by splitting the temporal muscle is the best. The dura is opened only if there is blood or accumulated fluid underneath, and is not drained unless it seems necessary. We agree with Heuer [20] and Bower [21] that subdural drainage should be avoided if possible because of the danger of infection, but it is definitely indicated at times and may save life. Exploration and drainage of a frontal sinus may be indicated if the fracture has probably torn the dura and extended into an infected sinus; this also applies to an aerocele from a fracture into the paranasal air cells. As a comparison to our experience, Sharpe [12] performed operations on 30 per cent; Stewart [4] on 28 per cent; Wilensky [22] on 24 per cent and Naffziger [23] on about 20 per cent of the patients, with mortalities ranging from 39 to 51.7 per cent. Operation was performed in twenty-two of our cases, or 4.3 per cent; fourteen patients died, giving an operative mortality of 59.1 per cent. In practically all of the fatal cases, it was found that extensive injuries were present besides that for which the operation was performed. This and the fact that so many patients whose condition was serious recovered by means of nonsurgical measures explains the operative mortality rate which is a little higher than in most of the other series. On the other hand, our mortality rate for the whole series was 14.7 per cent, which is the lowest of all except Hendon's [13] of sixty-three cases with 6 per cent and Munroe's [17] of ninety-nine cases with 14 per cent.

After-Care.—There can not be any hard and fast rules concerning the after-care of these patients, as each case presents a different problem. An essential in all is absolute rest for a sufficient period to assure return to normal and then a gradual return to active life. Our custom has been to keep patients in bed for from one to four weeks or longer; we then gradually permit them to be up and around during a period of from one to two weeks and instruct them to lead an easy life for from one to two months longer. In this way, they return to light work in from one to three months after injury and do regular heavy work after from one to three months more. Jackson,[9] Krause [24] and Sharpe [25] advocated

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20. Heuer, G. I.: Fracture of the Skull, J. A. M. A. **82**:1467 (May 3) 1924.

21. Bower, John O.: Management of Injuries to the Cranium and its Contents, Ann. Surg. **78**:433 (Oct.) 1923.

22. Wilensky: Fracture of Skull with Special Reference to Its Neurological Manifestations, Ann. Surg. **70**:404 (Oct.) 1919.

23. Naffziger, H. C.: Head Injuries—Indications for Surgical Treatments, S. Clin. N. Amer. **3**:699 (June) 1923.

24. Krause, Charles: Standardization of Treatment of Skull Fracture, J. Iowa M. Soc. **15**:181.

25. Sharpe, William: Recent Advances in Neuro-Surgery—Diagnosis and Treatment of Brain Injuries, Rhode Island M. J. **8**:51 (April) 1925.

an equally strict regimen. If headaches, vertigo or other unfavorable symptoms develop when the patient returns to work, a lumbar puncture may be indicated, and if the pressure is high, another period of rest is needed with a sufficient spinal drainage to return the pressure to normal. Administration of mild sedatives, such as phenobarbital may be necessary for nervousness or insomnia. One of the dangers in the cases is the development of neurotic fixations, particularly in certain types of compensation cases. Skilful handling is necessary to prevent this complication.

RESULTS

In reviewing a large series of old cases, it is impossible to arrive at a reliable classification of results. Some patients left the hospital early against advice; many did not return for a follow-up, and some doubtless

TABLE 7.—*Results*

Cured		Recovered, But With Sequelae on Discharge									Not Treated		Died					
Temporary Disabilities While in Hospital	No Disabilities or Sequelae	Headache	Vertigo	Stiffness	Cranial Nerve Involvement	Fatigue, General Disability	Nervousness	Psychoses	Epilepsy	Hemiplegia	Transferred to Another Hosp.	Left Against Advice	Less Than 6 Hrs.	7 to 12 Hours	13 to 18 Hours	19 to 24 Hours	2 Days	3 Days	Over 3 Days
39	244	27	2	1	11	21	3	8	4	1	6	9	28	10	3	2	10	6	6
Totals 283		78									15		65						
Percentage of total series																			
64.2		17.7									3.4		14.7						

left the hospital free from symptoms and developed unfavorable sequelae afterward, but did not return for observation. Table 7 shows our classification based on the patient's condition when he was discharged from the hospital. In 64.2 per cent of the cases, the patients were apparently cured and did not return with unfavorable symptoms; 17.7 per cent of the patients left with some unfavorable symptoms; 3.4 per cent were transferred or left against advice and 14.7 per cent died. We have tried to supplement this data by our follow-up notes and by a questionnaire sent out to the 229 who disappeared from observation. Of this latter group, 33.2 per cent replied; the letters sent to 21.9 per cent were returned undelivered and 44.9 per cent did not reply. Letters are still coming in from time to time. Of the seventy-six who answered the inquiry, 71.1 per cent had suffered or are still troubled with one or more unpleasant sequelae. These follow in the order of their frequency: vertigo, headaches, fatigue-ability, nervousness, irritability, impairment of memory, mental changes, paralyses and convulsions.

Sharpe [12] attempted such a survey and heard from 34 per cent of his patients; 68 per cent showed unfavorable symptoms. Hoag [26] stated that subjective symptoms are present in 80 per cent of the cases of injuries of the head, and that of these 8 per cent are psychic. When the compensation factor enters in, it is difficult to evaluate fairly the

TABLE 8.—*Cause of Death*

Head Injuries	Hemorrhage	Laceration	Laceration and Hemorrhage	Operation, Laceration and Hemorrhage	Meningitis	Pneumonia	Myocardial Failure	Totals
Not serious	..	..	..	1	..	1	1	3
Serious	2	11	35	9	4	1	..	62
Totals	2	11	35	10	4	2	1	65

TABLE 9.—*Our Summary and Others. Some in Recent Literature*

Author	Year	No. of Cases	Average Age	Trauma, %	Site %	Operations, %	Operative, Mortality, %	Total Mortality, %	Sequelae %
Besley	1916	1,000	42+	Fall 38 Blow 31	Vault..... 33 Base..... 34 Combined 33	..	1	53	
Sharpe	1916	239	..	.....	.....	..	..	30.7	67 of 34 return cases
Wilensky	1919	72	54	.....	.....	24	48	31	
Moorhead and Weller	1921	100	6½	.....	.....	12	..	26	
Stewart	1921	6,135 total 617 fracture skulls	36	Fall 33 Auto 24 Blow 22	Vault..... 66 Base..... 34	28	51.7	52	55
Bower	1923	62	..	.....	.....	..	..	26.1	
Brown and Strecker	1923	100	..	.....	Vault..... 47 Base..... 30	17	35.3	26	
Heuer	1924	223	..	.....	.....	23.8	41.5	35.8	
Hendon	1924	61 (one hosp.) 63 (another hosp.)			.....	20 10	40 50	60 6	
Connors	1925	1,100 possible	..	Series from 1914-1917		18.8	34.1	56.4	
		497 established cases	..	Series from 1917-1924		9.3	46.1	48.4	
Sharpe	1925	.....	..	.....	.....	30 (10% children)	39	33	
Munroe	1925	217 (other hospitals) 99 (his own)			....	3.2 10	57 33	22 14	
McClure and Crawford	1927	441	30	Auto 54.2 Fall 20 Blow 18.4	Vault..... 85.7 Base..... 5.7 Combined 8.6	4.3	59.1	14.7	17.7

organic and functional elements. Stewart [4] found 55 per cent with unfavorable sequelae in his series. In our series 81.5 per cent of the deaths occurred within forty-eight hours after admission. Table 8 shows the causes of death.

26. Hoag, David A.: Nervous and Mental Diseases Following Head Injuries, J. A. M. A. **82**:1468 (May 3) 1924.

For comparison, we have tabulated some of the data from other series which were suitable for comparison (table 9). The complications in our cases while in the hospital are shown in table 10. No doubt some cases of meningitis were prevented by prophylactic means, such as early drainage of frontal sinuses, debridement in compound fractures and aseptic treatment of the ears and nose and other parts. Harvey [27] reported remarkably good results with his serious cases by rigid adherence to aseptic principles.

Our cases of meningitis were all fatal in spite of every effort. Only the future will show whether or not we can develop a method of treatment to cure some of these patients. More radical early irrigation with specific serums and antiseptics seems to offer some hope.

The results of injuries of the head in children are somewhat more favorable. They seem to show fewer sequelae and have a greater per-

TABLE 10.—*Complications*

	Meningitis	Hemiplegia	Cranial Nerves	Hemorrhage	Abscess of the Brain	General: Pneumonia, Embolism, Cardiac Failure	Hernia Cerebri
Recovered in hospital.....	..	9	Third... 1 Fifth... 1 Seventh 3 Eighth. 1 Sixth... 1 — 7	..	1	1	1
Discharged with disability...	..	1	Fifth... 1 Sixth... 1 Seventh 4 Eighth. 8 —14				
Died.....	4	5	Eighth... 1	2	..	3	
Totals.....	4	15	22	2	1	4	1

centage of recovery. They, are, however, more apt to develop later sequelae such as epilepsy, spasticities, developmental retardations and other conditions. In 1927, Moorhead [28] reported 100 cases with a mortality rate of 26 per cent or 17 per cent if associated injuries were excluded.

PROGNOSIS

Prognosis should be guarded until at least six months or a year have passed. This is particularly true in children and in cases involving insurance and compensation. In our experience, even simple injuries may be seriously impaired later by organic or functional derangements. The return of the function of involvements of the cranial nerve is sometimes slow and distressing.

27. Harvey, Samuel C.: Compound Cranial-Cerebral Injuries, Boston M. & S. J. **23**:911 (Dec. 6) 1923.

28. Moorhead, J. J., and Weller, Walter: Fracture of the Skull in Children **74**:72 (Dec.) 1921.

SUMMARY

1. The problem of the proper management of cases of injuries to the head is of increasing importance. Much progress has been made, but an improvement can yet be made on our results.

2. It should be the aim of the medical profession to make more accurate diagnoses of cases so that the appropriate treatment can be applied to each different group. Only in this way can the number of deaths, complications and sequelae be cut down.

3. In the management of cases of injuries of the head stress is laid on: (1) the importance of accurate detailed history, (2) careful repeated examinations, (3) constant skilful nursing, (4) the taking of roentgenograms in all cases, (5) spinal punctures for diagnoses in all doubtful cases, when the patient is not in a state of shock and when block does not occur at the base of the cerebrum, and treatment with hypertonics to reduce cerebral compression, (6) properly timed surgical intervention for accessible hemorrhage, compound and depressed fractures and (7) insistence on keeping the patients quiet for a sufficient length of time and a gradual resumption of duties after recovery.

4. We have made a report on our series of 441 cases of craniocerebral injuries because we believe that such publication of critical reviews is of scientific value.

ARTERIOVENOUS ANEURYSM *

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In this discussion the term "arteriovenous aneurysm" is used to denote "any abnormal communication established between the arterial and venous channels" and includes, therefore, all direct and indirect communications variously spoken of as arteriovenous varix, varicose aneurysm, aneurysm by anastomosis, pulsating venous aneurysm and cirsoid aneurysm. These communications usually result from trauma to the vessels, such as piercing, incisive, lacerating, crushing or contusive wounds (cirsoid aneurysm of scalp is a notable example), and are known as acquired arteriovenous aneurysms; not infrequently they are produced by developmental anomalies, and are then termed congenital. The symptoms and systemic effects of the two types are essentially similar; the chief differences will be noted later.

In the Mayo Clinic from January, 1915, to April, 1926, operation was performed in forty-one cases of arterial and arteriovenous aneurysm. Twenty-five of these were arteriovenous; sixteen were acquired and nine were congenital.

In 1757, John Hunter first recognized and described the symptoms of arteriovenous fistula, and in 1920, Callander,[1] in an interesting report of a comprehensive study of the literature, recorded 447 cases, of which only three were congenital. While knowledge of the symptoms has been sufficient to enable one to diagnose the condition readily, and while operative technic has been developed so that accessible fistulas can be operated on successfully, surprisingly little was known of the pathologic physiology until five or six years ago. Not infrequently the dilatation of the artery proximal to the fistula and evidences of cardiac injury were observed, but the causal relationship between these observations and the fistula was not appreciated. For present knowledge credit is given especially to the studies of Matas,[2] Makins,[3] Halstead, Reid,[4] Holman,[5] Hoover and Beams,[6] and Lewis and Drury.[7]

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* From the Division of Surgery, Mayo Clinic.

* Read before the Pacific Northwest Medical Association, Boise, Idaho, June, 1927.

1. Callander, C. L.: Study of Arteriovenous Fistula with an Analysis of 447 Cases, Johns Hopkins Hosp. Rep. **19**:260, 1920.

2. Matas, Rudolph: Testing the Efficiency of the Collateral Circulation as a Preliminary to the Occlusion of the Great Surgical Arteries, J. A. M. A. **63**:1441 (Oct. 24) 1914; On the Systemic or Cardiovascular Effects of Arteriovenous Fistula, Tr. South. Surg. & Gynec. Assn. **36**:623, 1923.

3. Makins, G. H.: Gunshot Injuries to the Blood Vessels, New York, William Wood and Company, 1919.

(Footnotes continued on following page)

SYMPTOMS

The symptoms of arteriovenous aneurysm can be conveniently divided into local or regional, and cardiovascular or systemic.

Local Symptoms.—In the region of the fistula there is a continuous bruit and a purring thrill throughout the cardiac cycle with systolic intensification. These sounds are transmitted distally and proximally along the lines of the veins. The thrill is pathognomonic; it is present in all cases of arteriovenous aneurysms and is most marked at the site of the lesion. It is felt as a humming or buzzing sensation and has been likened to the vibrations of a bee in a paper bag. Often venous pulsations are visible, but this together with the murmur and thrill, disappear when the fistula is closed by digital pressure. A tumor may or may not be present. The superficial veins distal and proximal to the lesion are engorged and tortuous. In some of the cases in the present series, operation for varicose veins had been performed previously. The circumference of the limb is often greatly increased, and in the congenital cases the limb is actually lengthened. The surface temperature in the region of the fistula is increased from 2 to 3 C., while in the distal parts it is lower than that of its fellow. Blood taken from the veins near the site of the aneurysm is high in oxygen content, and may be equivalent to that of arterial blood. In the distal part of the limb there may be trophic changes such as gangrene and varicose ulcers. Owing to the venous engorgement, the patient may complain of weakness, of a feeling of heaviness and numbness and a sense of weight in the limb. In many instances an elastic bandage is necessary for relief.

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4. Reid, M. R.: Studies of Abnormal Arteriovenous Communications, Acquired and Congenital: I. Report of a Series of Cases, Arch. Surg. **10**:601 (March) 1925; Abnormal Arteriovenous Communications: II. The Origin and Nature of Arteriovenous Aneurysms, Cirsoid Aneurysms and Simple Angiomas, ibid. **10**:996 (May) 1925; Abnormal Arteriovenous Communications: III. The Effects of Abnormal Arteriovenous Communications on the Heart, Blood Vessels and Other Structures, ibid. **11**:25 (July) 1925; Abnormal Arteriovenous Communications: IV. The Treatment of Abnormal Arteriovenous Communications, **11**:237 (Aug.) 1925.

5. Holman, Emile: The Physiology of Arteriovenous Fistula, Arch. Surg. **7**: 64 (July) 1923; Arteriovenous Aneurysm, Ann. Surg. **80**:801, 1924; Experimental Studies in Arteriovenous Fistulas, Arch. Surg. **9**:822 (Nov.) 1924. Holman, Emile and Edwards, M. E.: A New Principle in the Surgery of the Large Vessels: Ligation of Vein Proximal to Site of Ligation of Artery; Experimental Studies, J. A. M. A. **88**:909 (March 19) 1927.

6. Hoover, C. F., and Beams, S. J.: The Diagnosis and Pathologic Physiology of Arteriovenous Aneurysm, Arch. Int. Med. **33**:1 (Jan.) 1924.

7. Lewis, T., and Drury, A. N.: Observations Relating to Arteriovenous Aneurisms; Circulatory Manifestations in Clinical Cases with Particulr Reference to Arterial Phenomenon of Aortic Regurgitation, Heart **10**:301, 1923.

Systemic Symptoms.—Systemic changes are not present in all cases of arteriovenous aneurysm; their presence or absence, as clearly proved by Holman, is dependent on two factors, the size of the fistula and the unobstructed return flow to the heart. The larger the opening and the longer it exists, the more likely the development of cardiovascular changes. Holman's explanation of the hydraulics of the flow of blood in cases of arteriovenous fistula, based on intensive clinical and experimental studies, is convincing. With the production of an arteriovenous fistula, there are two systems of circulating blood: the normal, from heart to artery to capillary bed to veins, and the abnormal, from heart to artery to fistula to vein. As the fistula offers considerably less resistance to the circulation of the blood than the capillary bed, a large volume of blood will be directed through this fistula, the amount varying with the size of the fistula. Only a relatively small volume of blood will escape through a small fistula, and there will not be any great change in the size of the vessels. If the opening is large, however, a greater volume of blood will flow toward and through it. The proximal artery and vein dilate to provide for this increased volume of circulatory fluid. As Holman has pointed out, "this dilatation is progressive and will continue until the resistance offered by the fistula, plus the resistance to any further dilatation of the vessels leading to it, equals the resistance which exists in the capillary bed elsewhere." The proximal artery becomes enormously dilated, and in places in which the artery is not firmly supported in a bed of muscles or fascia, the artery also is lengthened, as evidenced by its great tortuosity. I had an opportunity to explore the external iliac arteries in two cases of femoral arteriovenous aneurysm, and in both this bending and twisting was so marked that at least 20 per cent increase in length could be estimated. The shunting of this large volume of blood through the fistula increases the venous pressure, which causes the heart to fill more quickly, with a consequent increase in the force and the rate of action and ultimate hypertrophy and dilatation of the heart. This diversion of a large volume of blood from the normal system is followed by an immediate fall of general blood pressure. In order to maintain the blood pressure at a level compatible with health, there must be an increase in the total volume of circulating blood, in addition to the increased rate and force of the cardiac beat. Lewis and Drury believed that the enlargement of the heart associated with an arterial leak is due to dilatation and not to hypertrophy, and is the result of diminished flow of blood through the coronary arteries consequent to the marked lowering of the aortic pressure. As they could not determine any increase in the venous pressure in their cases, they concluded that the work of the heart was not increased, and therefore, its increased size after the establishment of an arteriovenous fistula was the result of

dilatation and not hypertrophy. Experimentally, Holman has shown that the total volume of circulating fluid is increased, that venous pressure is increased in the veins proximal to the lesion and that hypertrophy as well as dilatation of the heart results. In case 16, the total volume of blood before operation for closure of the fistula was 108 cc. for each kilogram of body weight, whereas after the operation it decreased to 85 cc., which is considered normal.

The effect on the cardiovascular system of an arteriovenous aneurysm is similar in some respects to that of aortic regurgitation. In the latter condition, however, the arterial leak is limited to diastole, whereas in a fistula the leakage occurs throughout the entire cardiac cycle. The systolic pressure is normal or slightly above normal, thus presenting a high pulse pressure, and the diastolic pressure is considerably below normal. The temporary closing of the fistula by digital pressure is followed by an immediate rise in systolic as well as diastolic pressure, the latter always more marked. The permanent closure of the fistula results in the restoration of the pressure to normal. The blood pressure in the limb distal to the anastomosis is obviously greatly diminished and below that of its fellow. The pulse is of the water-hammer type, and frequently capillary pulsation is visible. The heart beat is forceful, often heaving. Cardiac murmurs are frequently present.

The rate of the heart is usually increased. In the cases in this series of acquired arteriovenous fistulas in which any enlargement of the heart or of the proximal artery was noted, the heart rate was 64, 90, 62, 72, 92, 76 and 92. Closure of the fistula is followed by an immediate drop in rate, often as much as forty beats each minute (Branham bradycardia sign). This phenomenon is readily explained. The stoppage of the arterial leak restores to the circulation the normal resistance of the capillary bed, which, in face of the increased total blood volume produced in consequence of the compensatory mechanism to maintain general blood pressure in the presence of the circulatory short circuit, results in the sudden elevation of the arterial pressure with dilatation of the aorta. The vagal depressor fibers are thereby stimulated and an immediate slowing of the cardiac rate follows. That this is the result of vagal stimulation is conclusively proved by the abolition of the phenomenon if the patient has previously been given atropine for physiologic effect. When the fistula is closed permanently, the readjustment of the circulation, as evidenced by the gradual return of the blood pressure and the cardiac rate to normal is accomplished mainly by the diminution of the total blood volume.

There was definite evidence of cardiac change in nine of the twenty-five patients in this series.

The artery leading to the fistula is often enormously dilated, thin-walled, tortuous and heaving. In two cases of arteriovenous fistula of

the femoral vessels this dilatation extended up as far as the common iliac vessels. The proximal artery was dilated in nine of the sixteen cases of acquired aneurysm; it was normal in two, and the condition was not recorded in five. Dilatation was noted in three of the nine cases of congenital aneurysm. The frequent finding of calcareous plaques and the ease with which the vessels are ruptured indicate that the walls of the arteries are often degenerated as well as dilated. In case 8, a right popliteal arteriovenous aneurysm of fourteen years' duration and rupture of the right superficial femoral artery followed the application of a tourniquet. In case 15, a left femoral arteriovenous fistula of twenty-six years' duration, a large mass (from 15 to 20 cm. in diameter) developed in the left pelvis, and exploration revealed a retroperitoneal cystic mass, nonpulsating and containing old partially clotted blood. The examination of the wall of the mass showed the structure of an arterial wall, but a direct communication with the iliac artery could not be demonstrated. The degenerative process obviously results from the tremendous dilatation of the artery, which in turn prevents adequate nutrition of its walls by partially obstructing the vasa vasorum. The veins in the vicinity of the lesion are markedly dilated, also, and in cases of congenital lesions the walls of the veins are thickened, or arteriolized; in many instances it is difficult to differentiate the artery from the vein.

DIAGNOSIS

The diagnosis of arteriovenous aneurysm should not offer any difficulties. The characteristic bruit and thrill, continuous throughout the cardiac cycle with systolic intensifications, and the transmission proximally and distally along the line of the companion vein and its tributaries alone should make the diagnosis almost certain. An absolute diagnosis can be made if an analysis of the blood taken from one of the veins in the immediate vicinity of the lesion is found to be rich in oxygen.

PROGNOSIS

Although instances are reported in the literature of the spontaneous healing of recently acquired (within two or three months) arteriovenous aneurysms, it is extremely unlikely that any arteriovenous fistula of three or four months' duration will heal without surgical intervention. It is much more likely that the lesion will remain chronic and that the symptoms will progress steadily with enlargement of the fistulous tract. The ultimate prognosis of any untreated arteriovenous fistula will depend on the size of the fistulous opening, the most influential factor in the production of cardiovascular injury. Reid reported that Osler for years followed two cases of arteriovenous aneurysm (one axillary and one femoral) in which death occurred from cardiac disease at the early ages

of 29 and 46, respectively. Osler did not appreciate the causal relationship between the two conditions, and counselled against operation in cases of arteriovenous aneurysm. The attitude formerly held by the medical profession, based on the chronicity and often apparent absence of symptoms, that communication between one of the smaller arteries and veins is a relatively benign lesion, has been materially changed, so that today the cardiac injury, chronic invalidism and the shortening of life, are recognized as potential complications of the lesion.

TREATMENT

The object of any surgical treatment in cases of arteriovenous aneurysm is to stop permanently the arterial leak without interference with the blood supply of the tissues beyond. The method of accomplishing this may vary according to the type and site of the lesion, the efficiency of the collateral circulation, and the individual predilection of the surgeon.

Congenital Arteriovenous Aneurysm.—Reid reported a case of congenital arteriovenous aneurysm in which operation revealed one communication between the subclavian artery and vein, although the application of ten ligatures was necessary for cure. In most cases, however, of this kind there are multiple communications between the arteries and veins, and often between the smaller branches. The treatment, therefore, must be directed at the elimination of the mass of dilated vessels and their communications as in cases of superficial cirsoid aneurysm, or the affected limb must be amputated. In three cases in the series in which a limb was involved, a maximal thrill could be felt over one of the larger arteries, suggesting that the communication was single; operation was advised with a view of extirpating the fistula. After the vessels were exposed a definite area of anastomosis between the artery and the vein was found and removed, but following this procedure in each case the circulation in the distal parts was definitely diminished; gangrene resulted and amputation of the limb became necessary. Subsequent radiologic examination and dissection of these amputated limbs showed that numerous communications between the arteries and veins were scattered diffusely through the limb. Many of the communications were only minute; their presence precluded the possibility of any effectual surgical treatment other than amputation.

Acquired Arteriovenous Aneurysm.—In the acquired form of aneurysm there is usually only one communication, and the operative procedure for the extirpation is relatively simple. Unless a pulsating hematoma accompanies the fistula, or the wound is badly infected, it is advisable to wait two or three months before attempting repair. This delay permits healing of the wound and subsidence of the edema of the

tissue, the establishment of the collateral circulation and sufficient opportunity for spontaneous healing of the fistula. The collateral circulation in all the cases of this series was adequate; indeed, Reid asserted that the presence of a fistula acts as a powerful stimulus to the development of a collateral circulation. A preliminary test (such as Matas') of the efficiency of the collateral circulation is indicated.

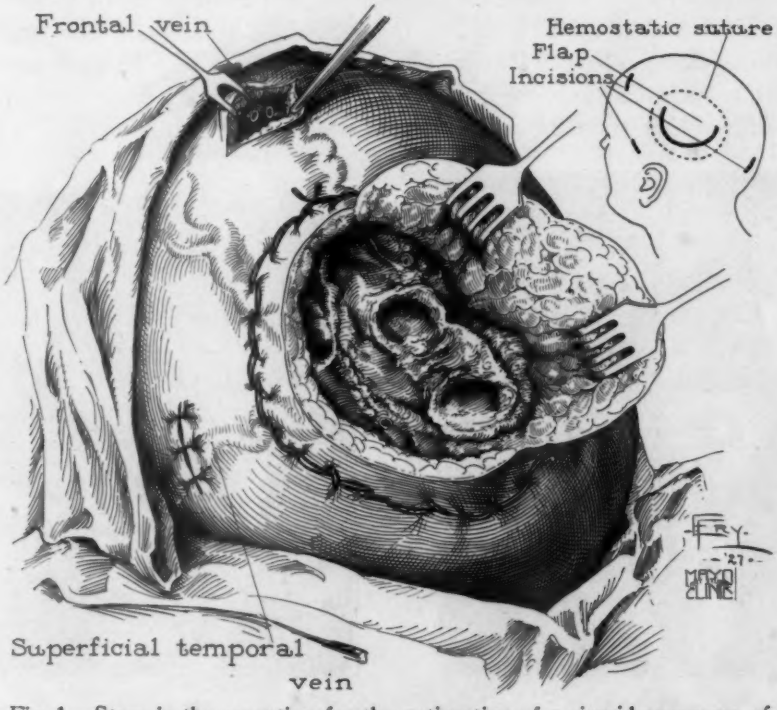

Fig. 1.—Steps in the operation for the extirpation of a cirsoid aneurysm of the scalp. Several of the pulsating vessels leading to the aneurysmal mass are ligated through small separate incisions, usually including both superficial temporal artery and vein, the occipital artery and vein, and the supraorbital artery and frontal vein; a continuous locked suture (hemostatic) is placed in the scalp around the periphery of the aneurysm; a semicircular incision is made through the scalp within the circle of the hemostatic stitch, and the scalp flap over the aneurysm is reflected, the mass of dilated vessels is extirpated by exeision; open vessels are tied and sutured; the hemostatic circular suture cut, a segment at a time, and further bleeding in the wound controlled by suture; the flap is replaced and edges of the wound closed.

Operative Procedures.—One of four operative procedures may be employed in the treatment of arteriovenous aneurysm:

1. A segment of the artery and vein, including the fistulous tract, may be excised. This is the surest method of permanently obliterating the communication, and is attended by little risk. I believe that this

should be the procedure of choice in cases in which it is known that the collateral circulation is efficiently established.

2. The fistulous tract may be ligated with heavy silk. This is the simplest form of treatment and is admirably suitable in cases in which the vessels can be mobilized and the communicating tract readily isolated. It is especially applicable in cases of carotid jugular fistula.

3. Restorative angiorrhaphy should be employed in cases in which doubt exists as to the efficiency of the collateral circulation. This operation may be accomplished by one of several methods: (*a*) In the presence of an aneurysmal varix the fistulous tract may be divided between clamps and the openings in the artery and vein closed by a running suture of

Fig. 2A Fig. 2B

Fig. 2.—Traumatic pulsating exophthalmos of six weeks' duration; *A* shows the patient before operation; *B*, seven days after ligation of the left internal carotid artery.

silk; this method is applicable only in cases in which both the artery and vein in the region of the anastomosis can be easily mobilized. (*b*) Approach to the fistula may be made by incising the wall of the vein opposite the point of anastomosis (Bickham-Matas operation) and the orifice of the fistula sutured from within the vein, the lumen of the vein may then be reestablished by closure of the incision as a lateral phleborrhaphy, or the vein may be occluded and its walls sutured to the artery so as to reinforce the line of closure. (*c*) In case of varicose aneurysm the approach for suturing the openings in the vein and artery may be made through the aneurysmal sac, as an endo-aneurysmorrhaphy (Matas). The chief advantage of the operation of angiorrhaphy is that the circula-

tion to distal parts is not endangered; the principal objections to its employment is the danger of secondary hemorrhage. Theoretically, this is more likely to follow suturing of an artery in the presence of arteriovenous fistula because of the greater possibility of faulty union, owing to the increased blood pressure and the associated dilatation and degenerative changes in the wall of the artery.

4. The ligation with silk of the artery and vein proximal and distal to the anastomosis (quadruple ligations) has proved successful in many cases; its chief objection is that the circulation through the large tied vessel is sometimes reestablished subsequently either by loosening of the sutures or by spontaneous severance of the wall of the vessel by the constricting suture with complete union of the ends of the vessel (fig. 1).

TABLE 1.—*Effect of a Femoral Arteriovenous Aneurysm on the Circulation and on the Measurements of the Limb*

	Fistula Open	Fistula Closed	Before Operation	After Operation
Blood Pressure				
Systolic	140	185	140	120
Diastolic	40	80	40	80
Heart rate	90	60	90	80
Blood volume			108 cc. for each kilogram of body weight	85 cc. for each kilogram of body weight
Oxygen content, venous blood			18 volumes per cent	
Size of heart			12 cm. to left in fifth intercostal space	9.5 cm. to left in fifth intercostal space
Temperature				
Left thigh			30.2 C.	34.4 C.
Right thigh			33.3 C.	
Circumference				
Left thigh			53 cm.	45 cm.
Left calf			36.5 cm.	31 cm.
Right thigh			42 cm.	42.5 cm.
Right calf			29.5 cm.	28 cm.

The operation consisting of ligating the proximal artery only is obviously definitely contraindicated in all arteriovenous aneurysms, except those within the cranium, such as pulsating exophthalmos. If the proximal artery of an arteriovenous aneurysm involving trunk vessels of an extremity is ligated, then the blood supplied to the parts distal to the anastomosis by way of the arterial collaterals will flow in the direction of least resistance, i.e., in a retrograde manner through the distal artery by way of the fistulous opening back to the heart. The greater part of the collateral circulation will accordingly be shunted back to the heart without supplying adequate nutrition to the extremity. Trophic sores and gangrene become inevitable.

In cases of intracranial arteriovenous fistula, direct attack on the fistula is impossible and experience has proved that in a great many of these cases in which the internal carotid and the cavernous sinus (pulsating exophthalmos) particularly those of traumatic origin are involved, the patients are cured by proximal ligation of the internal

carotid (fig. 2). In such cases the communicating tract must be very small to favor closure by clotting. But even so it is difficult to understand how cure is effected unless the slowing of the blood stream through the fistula incident to the ligation of the internal carotid artery is more marked than usual. This might be caused by the additional obstruction to the flow of blood offered by the trabeculations in the cavernous sinus which permits clotting in the fistulous tract before the secondary rise in blood pressure in the carotid after the complete reestablishment of the collateral circulation.

Fig. 3 (case 16).—Arteriovenous aneurysm of the left common femoral vessel of six years' duration. The prominence of the tortuous superficial veins, the increased dimensions of the limb, and the presence of the trophic ulcer of the leg may be noted.

One of the most important principles in surgery of the vascular system was not recognized until at the time of the World War, when Sir George Makin pointed out that in case of injury to an important artery the simultaneous ligation of both artery and vein does not give rise to increased risk of gangrene, but diminishes it. By obstructing the companion vein also the balance of the collateral circulation is more

efficiently maintained. In case the artery alone is ligated, the blood supplied by the arterial collaterals finds a too ready exit through the unobstructed vein. By ligating the vein also, the blood pressure in the distal parts is actually increased. This has been substantiated by ample experimental and clinical observations. Recently, Holman and Beam have shown by experiments on the dog that the blood pressure in an extremity in which the main artery has been ligated will be materially elevated if the satellite vein is obstructed at a corresponding site, and, if the obstruction is made at a point proximal to the ligature on the artery so as to include the important venous tributaries, the pressure will be raised to an even higher level.

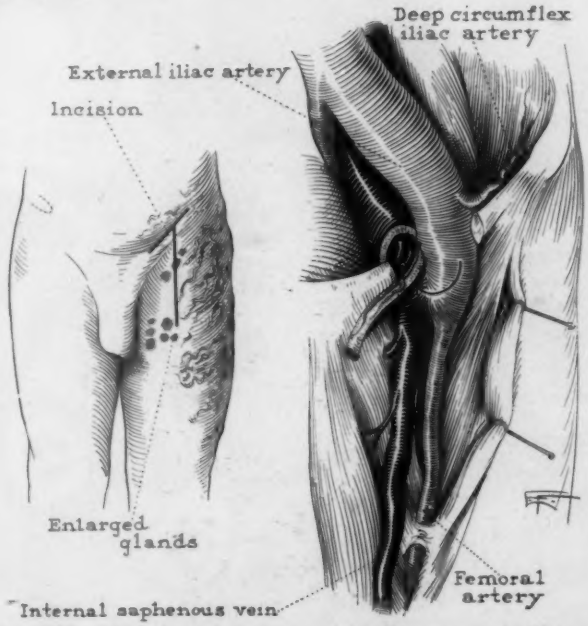

Fig. 4 (case 16).—Arteriovenous aneurysm. The arrow indicates the position of the fistula. The enormous dilatation of the proximal artery and vein with diminution of the size of the distal artery were noted at operation.

REPORT OF CASES OF ACQUIRED ARTERIOVENOUS ANEURYSM

CASE 1 (case 16, table 2).—*History.*—A man, aged 19, was admitted to the Mayo Clinic, Feb. 22, 1926, complaining of a swelling in the left leg, so severe that his local physician had advised him to wear an elastic stocking. About six years previously, he had been accidentally wounded with a 22-caliber rifle. The bullet entered the anterior medial surface of the left thigh, about 13 cm. below the inguinal ligament. The bullet was not removed. There was considerable hemorrhage at the time of the accident and the patient fainted. He said that he felt a thrill in the upper part of the thigh before leaving the hospital; the thrill increased progressively. In a few months he noticed that the left thigh and leg were larger than the right; also that the veins in the left leg had become distended. Three years later he injured the anterior surface of the left leg and an ulcer appeared that did not heal.

Examination.—The patient weighed 69 Kg. (131 pounds). There was a marked systolic pulsation in the vessels of the neck with slight systolic thrill and murmur over the carotids. Cardiac dulness extended 12 cm. to the left and 2 cm. to the right of the median line, with a precordial heave from the second rib down and a systolic murmur over the whole precordium. The point of maximal impulse was heaving and powerful. The superficial veins of the left side of the abdomen

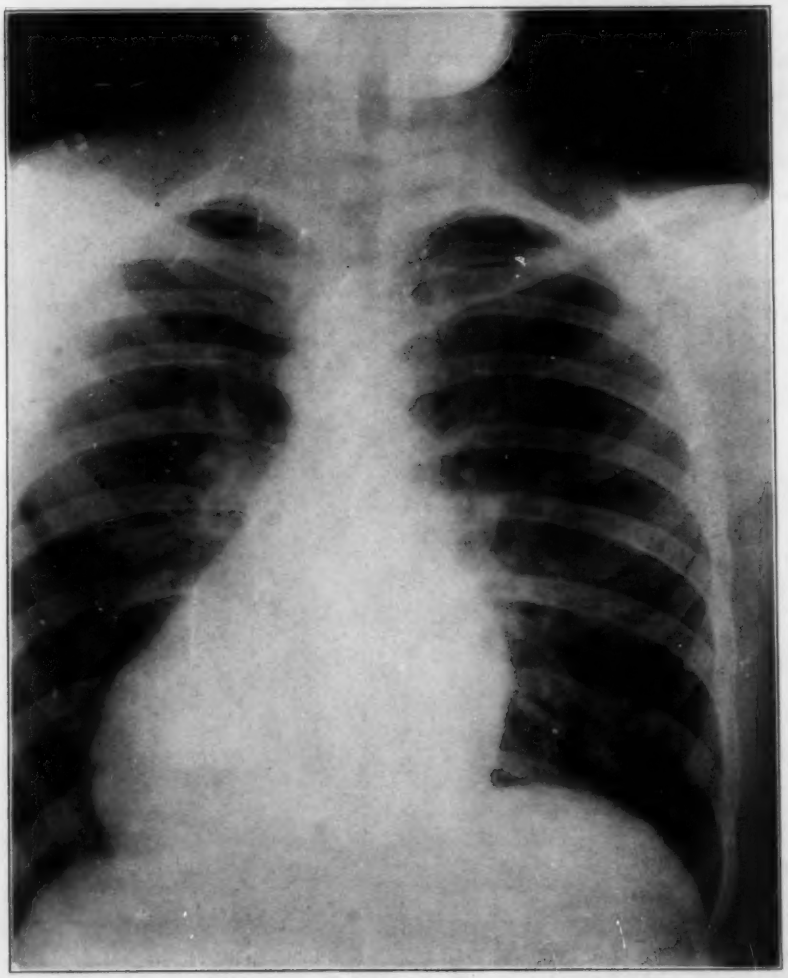

Fig. 5A

Fig. 5 (case 16).—*A* shows the heart before operation; *B*, thirty-one days after operation.

and the left inguinal region were dilated and could be followed to the left side of the chest. A continuous thrill with systolic intensification could be felt over the left Poupart ligament. Capillary pulsation was observed in the fingers. The right thigh measured 42 cm., and the left, 53 cm. The right calf measured 29.5 cm., and the left, 36.5 cm. The left leg was warmer than the right, more

marked in the thigh. All veins of the left leg were distended. The blood pressure with the fistula open was 142 systolic and 40 diastolic, and with the fistula closed, 164 systolic and 80 diastolic. There were partly healed ulcers on the anteromesial surface of the left leg. Two roentgenograms were taken, one of the chest which showed the heart to be enlarged 2, the other of the left hip which showed a large fragment of lead over the head of the femur with small particles over the neck

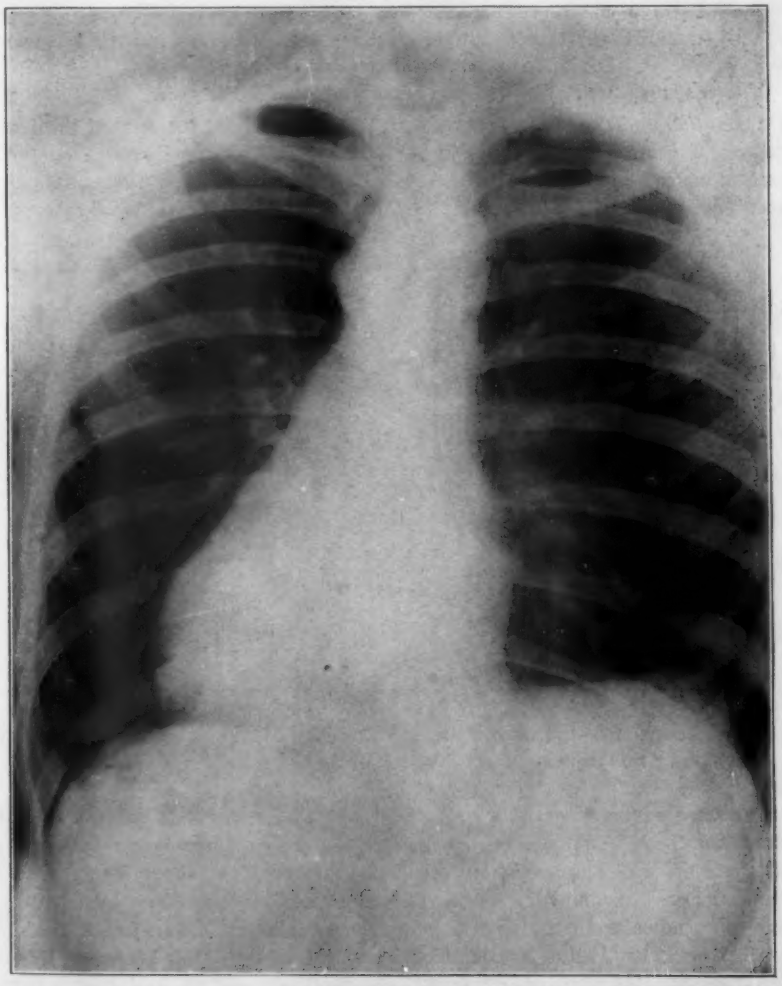

Fig. 5B

and inner aspect above the trochanter. The electrocardiographic report showed a pulse rate of 86; sinus rhythm; aberrant ventricular complex in isolated derivation I, and slurred right ventricular preponderance. The systolic blood pressure was 150, and the diastolic was 60. The hematocrit showed that the plasma was 65 per cent and the erythrocytes, 35 per cent. The oxygen capacity was 23.2 per cent, and the oxygen content, 18.2 per cent, making an oxygen saturation of 78 per cent. The carbon dioxide combining power was 66 per cent by

volume. The plasma volume for each kilogram was 70 cc., the total volume being 4,857 cc. The whole blood volume for each kilogram of body weight was 108 cc. (normal 85), the total volume was 7,462 cc. Subsequently, the blood pressure of the left leg with the fistula open was 140 systolic and 40 diastolic; with the fistula closed it was 185 systolic and 80 diastolic. The pulse rate was 90 with the fistula open and 60 with the fistula closed. The temperature of the left thigh was 3 C. warmer than the right. The oxygen content of the venous blood of the left thigh was 18 volumes per cent, equal to the arterial blood; its oxygen capacity was 23.2 per cent by volume. The blood volume for each kilogram of body weight was 108 cc.; the total volume was 7,462 cc.

The diagnosis was arteriovenous aneurysm of the left groin.

Operation and Course.—On March 1, 1926, I excised a segment of the femoral artery and vein, including the fistula, and placed quadruple ligatures on the left femoral artery immediately below Poupart's ligament. The first incision was made just above and parallel to Poupart's ligament. The external iliac artery

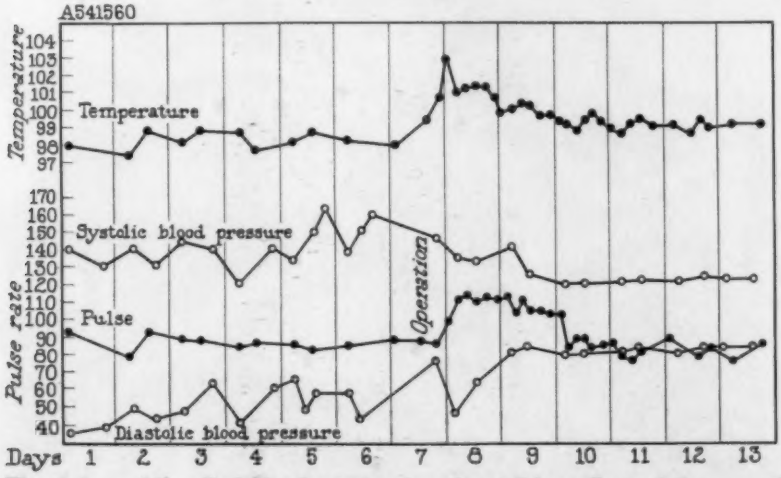

Fig. 6 (case 16).—Bedside chart showing the striking effect of the operation on the blood pressure.

was exposed extraperitoneally and tape was placed around it for control. The artery was 3 or 4 cm. in diameter, being four or five times larger than normal, and the vein was hugely dilated, probably two or three times larger than the artery; marked thrills were felt. A vertical incision was then made over the thigh, perpendicular to the first, running over the anterior surface of the thigh, about 12 or 15 cm. below the level of Poupart's ligament. The femoral vessels were exposed. The superficial veins were markedly dilated and bled profusely when opened. The femoral artery below the anastomosis was small, about one-fourth the size of the external iliac. A direct communication between the artery and the vein could not be made out because of the excessive amount of scar tissue. I finally tore into the artery and ligated it, and then removed a segment of the artery and vein, including the origin of the profunda femoris. The segments of the artery and the vein were probably 7 or 8 cm. in length. Both ends of the artery and vein were tied with heavy fish-line silk. The operation was extremely difficult on account of the situation. In order to secure exposure it was necessary to cut Poupart's ligament. This was later resutured. Lymph nodes in the

inguinal region were greatly enlarged and it was necessary to remove some of these in order to obtain exposure. The internal saphenous vein was ligated, and the external iliac was doubly ligated just above Poupart's ligament. The deep circumflex iliac and the deep epigastric arteries were also ligated. Two Penrose drains were inserted, one in each wound. Convalescence was normal. On the third day after the operation the pulse was dicrotic. The size of the heart was diminished.

On March 7, the right thigh measured 42 cm., and the left, 48 cm.; the right calf measured 29 cm., and the left, 31 cm. The systolic blood pressure was 120;

Fig. 7 (case 16).—Thirty-one days after operation for excision of fistula. A letter from the patient in January, 1927, stated that his general condition was excellent.

the diastolic, 80. On March 21, the right thigh measured 42.5 cm.; the left, 45 cm.; the right calf measured 28 cm., and the left, 31 cm. On April 3, the systolic blood pressure was 115; the diastolic, 80. The heart was reduced in size; there were no murmurs and the action was regular. On April 19, the patient was dismissed. Both wounds in the operative field and the ulcers were healed. Above the left Poupart ligament the tissues beneath the skin were indurated, and formed an irregular mass probably from 8 to 10 cm. in diameter. This induration could be felt with the finger in the rectum. There was no soreness at the point of induration and no pulsation; the induration was attributed

to the silk suture. The patient's general condition was good and the pulse was normal. There was practically no swelling in the leg. The result was excellent.

After operation the patient weighed 53 Kg. (117 pounds), and the hematocrit showed 34 per cent erythrocytes and 66 per cent plasma. The whole blood volume was 85 cc. for each kilogram of body weight (normal 85); the total volume was 4,515 cc. The plasma volume was 56 cc. for each kilogram of body weight, the total volume was 2,980 cc. (figs. 3 to 7 and table 1).

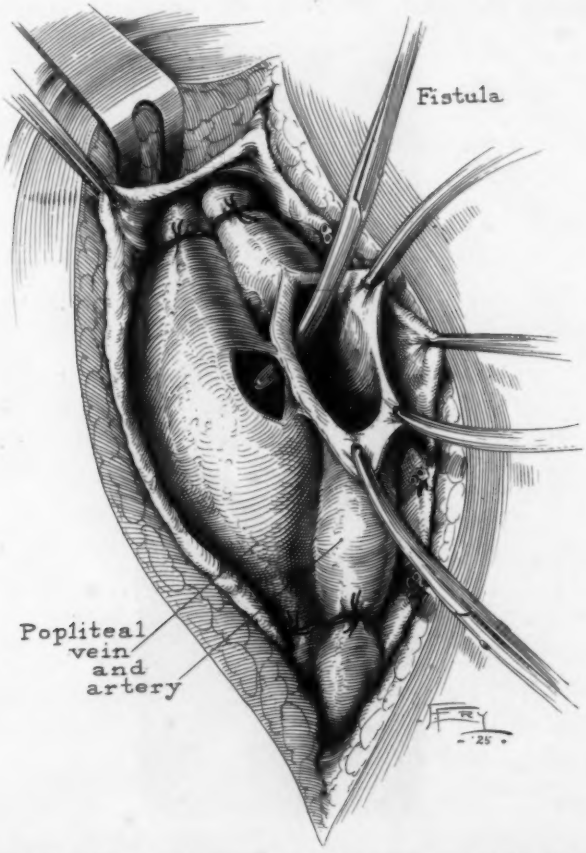

Fig. 8 (case 15).—Traumatic arteriovenous aneurysm of the left superficial femoral artery and vein of twenty-six years' duration. Quadruple ligations preliminary to the excision of segments of artery and vein, including the fistulous tract.

Case 2 (case 15, table 2).—*History.*—A man, aged 39, came to the clinic on Nov. 17, 1919, complaining of varicose veins which had first appeared at the age of 15. He gave a history of a gunshot injury at the age of 13, at which time a 22-caliber rifle bullet entered the left thigh. Enlargement of the veins of the left leg appeared one year later and arteriovenous aneurysm one year after this. Operation was performed when he was 18, and part of the veins were removed; however, they soon reappeared. The aneurysm had grown larger gradually,

and there was some edema of the leg. An ulcer just above the ankle had existed since he was 15; this had gradually increased in size.

Examination.—An aneurysmal mass in the middle third of the thigh posteriorly, and a large ulcer of the left leg were present. The systolic blood pressure was 115; the diastolic, 65. There was a systolic murmur to the left and over the precordium; the heart was enlarged to the left and downward, and the pulmonic second sound was accentuated. Over the left femoral artery was a to-and-fro systolic murmur and a bruit was transmitted upward and downward from the aneurysm. No anterior or posterior tibial pulse was felt. The results of urinalysis were negative, except for pus cells, grade 4. The combined phenolsulphon-

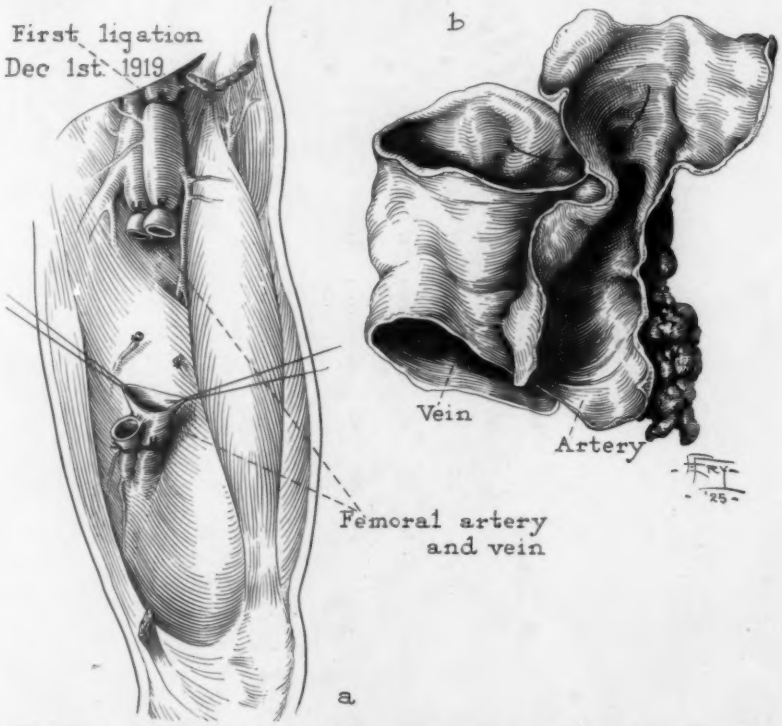

Fig. 9 (case 15).—Excision of segments of the superficial artery and vein, including the fistula. Six years previously the first part of the left femoral artery and of the fistulous tract had been ligated with heavy silk with temporary improvement. Circulation was subsequently reestablished at site of both ligatures and was followed by the development of a deep and exceedingly foul smelling trophic sore of the leg. Immediately after excision of the fistula marked improvement in the circulation of the foot and leg with rapid healing of the ulcer was noted.

phthalein test was 20 per cent in two hours. The blood urea was 16 mg. per hundred cubic centimeters. Roentgenogram of the kidneys, ureters and bladder was normal. Cystoscopic examination revealed a stricture of the bulbous urethra.

Operation and Course.—Operation was performed on Dec. 1, 1919. There was a large fusiform dilatation of the entire femoral artery from beneath Poupart's

TABLE 2.—*Acquired Arteriovenous Aneurysms* *

Case	Age, Years	Sex†	Cause	Lesion	Duration, Years	Cardiac Enlargement	Pulse	Blood Pressure: Systolic	Blood Pressure: Diastolic	Trophic Changes	Date of Operation	Type of Operation	Proximal Artery Dilated, Grade	Result	Comment and Subsequent History
1	29	♂	Bullet	Arteriovenous aneurysm of left popliteal vessels	11	.....	70	134	82	Varicose veins and scars of previous operation	9/25/15	Endo-aneurysmorrhaphy	—	?	Letter May, 1916: Sore on leg; numbness, thrill before bending leg; flexion impeded
2	49	♂	Lacerated wound	Arteriovenous aneurysm of right palmar arch	42	.....	88	138	90	Gangrene of finger	5/29/16	Partial amputation of right hand	..	Well	
3	35	♂	Bullet	Arteriovenous aneurysm of left subclavian	9	Slight (roentgenogram)	64	120	66	Tremendous congestion	5/23/16	Obliteration of fistulous tract by ligation	+	Well	Letter March, 1926: Slight swelling; strength all right; examination by home physician; condition satisfactory
4	60	♂	Blow	Cirsoid aneurysm of forehead	42	.....	..	142	80	.....	1/25/18	Ligation of vessels.....	..	Improved ?	
5	38	♂	Bullet	Arteriovenous aneurysm of left popliteal	1¼	.....	76	118	72	Tremendous congestion	6/ 6/18	Excision and ligation..	..	Well	Letter March, 1926: well
6 (figs. 9 to 12)	46	♂	Bullet	Arteriovenous aneurysm of right jugular-carotid vessels	30	+	90	120	64	.....	7/26/19	Ligation of communicating fistulous tract; small sacular aneurysm of carotid artery opposite site of arteriovenous fistula; neck ligated	2	Well	Died Dec. 9, 1926, following operation elsewhere for cancer of the stomach
7	29	♂	Bullet	Arteriovenous aneurysm of left peroneal	15	?	88	140	75	Ulcers of left leg	6/27/19	Excision and ligation of artery and veins	..	Well	Letter May, 1926: well
8	31	♂	Bullet	Arteriovenous aneurysm of right popliteal vessels	14	.....	62	134	60	Engorgement of veins of leg	9/27/19	Ligation with catgut of communication between artery and vein	4	Died	Secondary hemorrhage six days after operation; second operation for excision of aneurysmal sac; dry gangrene of foot and lower leg; rupture of right femoral artery result of injury from tourniquet; necropsy: huge dilatation of right common and external iliac and right femoral arteries

9	21	♂	Bullet	Arteriovenous varix between right carotid and internal jugular veins	6 (roentgenogram)	+	72	152	58	.....	12/3/19	Ligation of communicating branch; small saccular knob (aneurysm) 2.5 cm. on anterior surface of internal carotid at site of fistula; neck ligated	3+	Well	Last report two years ago
10	21	♂	Bullet	Arteriovenous aneurysm of right axillary vessels	⅝	.....	80	120	70	Tremendous congestion of arms	5/ 6/20	Endo-aneurysmorrhaphy	+	Well	Brachial plexus involved in aneurysmal mass and accounted for atrophy of hand and forearm; last report, December, 1921
11	31	♂	Bullet	Arteriovenous aneurysm of left femoral vessels	2½	Marked	92	140	50	Marked varicose veins of leg	2/ 2/21	Ligation of communicating fistula	3+	Improved	Slight thrill and bruit, but condition improved 90 per cent; under periodic observation
12	30	♀	Puncture wound with scissors	Arteriovenous aneurysm of right posterior tibial	20	.....	70	108	70	Varicose veins of legs; scars of previous operation	4/21/21	Ligation of artery and veins above and below fistula	—	Well	Letter, May, 1926; "Wonderful success of operation"
13	48	♂	Blow to jaw	Aneurysm of right facial artery and vein	2	—	72	115	78	.....	8/ 8/21	Excision and ligation..	—	Well	Letter, March, 1926
14	46	♂	Contusion from fall	Cirsoid aneurysm of scalp frontal region	24	—	88	132	88	.....	8/27/24	Ligation of temporal arteries and excision of aneurysm	2	Well	Last report, January, 1925
15‡	39	♂	Bullet	Arteriovenous aneurysm of left femoral artery at apex of Scarpa's triangle	26	+	..	115	65	Deep ulcer on leg	12/1/19	Proximal ligation of artery just beneath Poupart's; ligation of communicating branch	2	Improved	Worked as cook until readmission, November, 1925
						+ Extra systole	76	122	60	Deep sloughing ulcer	11/4/25	Excision and ligation of femoral arteries and veins	3	Well	Pelvic mass 15 to 20 cm. in diameter, slightly to left of midline discovered during convalescence from second operation; cystic clinically, questionable expansile pulsation; encysted hematoma, origin questionable; recent report: condition very good
16‡	19	♂	Bullet	Arteriovenous aneurysm left femoral just beneath Poupart's ligament	6	Dilatation 2+	92	164	80	Ulcer of leg	3/ 1/26	Excision and ligation	2+	Well	Letter from patient January, 1927: general condition excellent; slight serous drainage from abdominal wound

* Pemberton, J. de J., and Saint, J. H.: Congenital arteriovenous communications, Surg., Gynec. & Obst. (in press).
† In this table, ♂ indicates male; ♀, female.
‡ Reported in text.

ligament to about 9 cm. from the popliteal. For 10 or 12 cm. above the knee immediately below the apex of Scarpa's triangle, the artery was more distended than elsewhere, and there seemed to be a constriction just above this. The artery was tightly adherent to the vein at the point of constriction, and it is possible that there was a communication between the artery and the vein at this point, although it could not be definitely made out. The artery was separated from the vein high in Scarpa's triangle and a tape placed around it to control bleeding. The artery was injured at this time and there was some bleeding which was easily controlled by digital pressure. Below this point the artery was separated from the vein above and below the constriction, near the site of the bullet wound, and a silk ligature was passed around the point of possible communication with the vein.

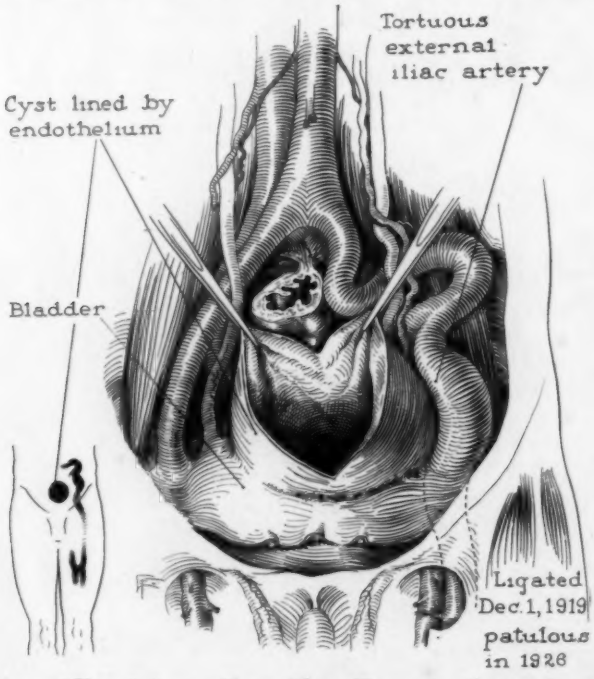

Fig. 10 (case 15).—A large (from 15 to 20 cm. in diameter) retroperitoneal cystic tumor lying slightly to the left of the median line and above the bladder, lined by endothelium and containing old, partly clotted blood was first discovered during the convalescent period after the excision of the fistula. The origin or the exact nature of the cyst could not be determined, but any connection with the iliac arteries was definitely excluded.

This, of course, produced further contriction of the artery. A definite purring thrill could not be felt in the vein before this ligation, but afterward there was less impulse. On removal of the tape, there was considerable bleeding from the artery, which could not be controlled other than by ligating the femoral artery just below Poupart's ligament and above the deep profunda artery. After the ligation there was a definite pulsation in the distal artery (dilatation of about 3 cm. in diameter) which showed that the collateral circulation was well established.

At operation on December 24, Thiersch's skin graft was applied to the ulcerated area of the left leg. Convalescence was uneventful. The patient was dismissed on Feb. 11, 1920, with wound healed, except for a varicose ulcer.

Second Admission to the Hospital.—The patient returned on Oct. 28, 1925, with a history of having been more or less incapacitated since the operation in 1919. He did not work for a year and a half after the operation and then worked as a cook in a construction camp for about two years. During the last summer, his left leg had given more trouble than before the operation.

On admission there was a deep malodorous sloughing sore above the left ankle, and the foot was bluish and cool. There was fusiform dilatation of the left femoral artery which extended from the left external iliac, 5 cm. above Poupart's ligament to the popliteal region. Thrills and bruits were present throughout most of this area and there were many varicose veins and much scarring. The knee could not be completely extended. The results of urinalysis were negative, except for pus, grade 4. The hemoglobin content was 74 per cent, the erythrocytes

Fig. 11 (case 6).—Traumatic arteriovenous aneurysm of the right jugular carotid vessels of thirty years' duration.

numbered 4,350,000 and the leukocytes, 12,900. The blood Wassermann test was negative. The phenolsulphonphthalein return was 50 per cent. The blood urea was 16 mg. per hundred cubic centimeters. A roentgenogram of the chest was negative. The same cardiac conditions were manifest, but there were occasional extrasystoles. The systolic blood pressure was 122, and the diastolic, 60.

Second Operation.—On April 11, an incision was made in the scar of the old incision. There were a great many adhesions between the artery and the muscles, so many that it was difficult to free the artery above this point, and profuse bleeding from the collateral veins was encountered, the blood being arterial in character. This was controlled with great difficulty, but I finally succeeded in putting a tie of heavy silk around the vessel above this point, including both the artery and the vein. This did not check the bleeding, and it was necessary to break up the adhesions hastily and place a mass tie around both the artery and the femoral vein below the aneurysm. This stopped the bleeding. The artery was then laid open, disclosing a small fistula about 1 cm. in diameter between the artery and the vein, and enormous dilatation of the vein. The femoral artery and vein were ligated en masse and separately above and below the aneurysm. Good

pulsation was noted in the distal end of the femoral artery after the completion of the operation, showing that the collateral circulation was well established. A segment of the artery and vein, including the arteriovenous fistula about 4 or 5 cm. in diameter, was removed. The wound was only partially closed and packed with gauze soaked in mercurochrome-220 soluble. It was necessary to give a transfusion of 500 cc. of blood while the patient was on the operating table. The patient's condition was good when he left the table. A similar transfusion

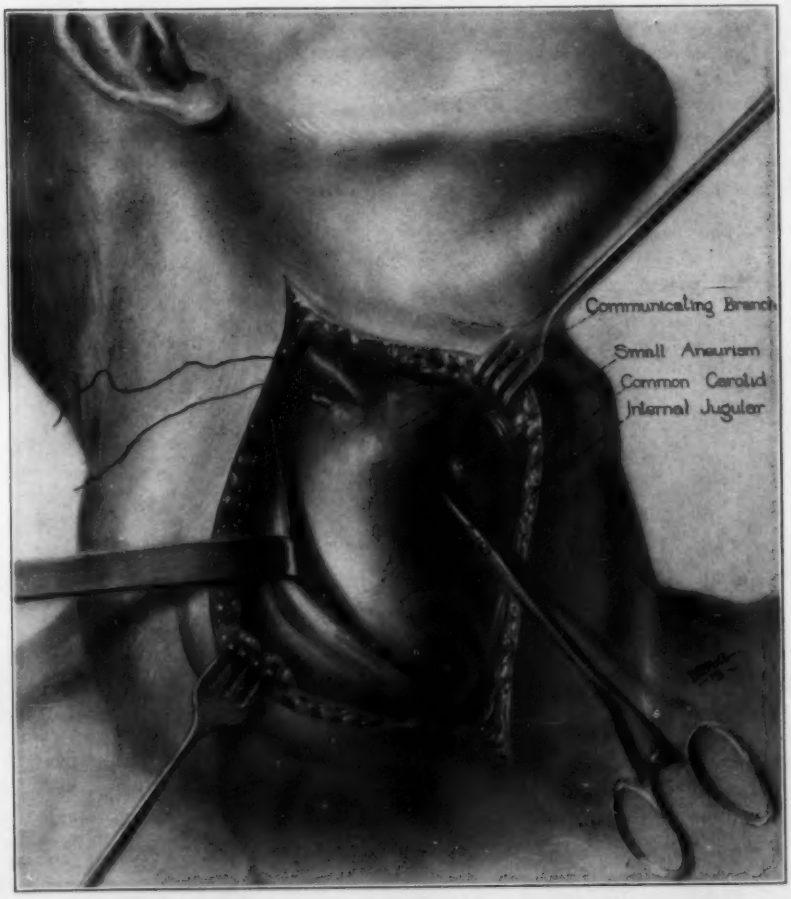

Fig. 12 (case 6).—Dissection exposing the enormously dilated internal jugular vein, the common carotid artery, and isolation of the communicating branch.

was given in the afternoon of the day of the operation. The pathologic report was arteriovenous aneurysm, with arteriovenous fistula about 5 mm. in diameter.

On November 8, there was an indefinite mass over the left Poupart ligament, which felt expansile. On November 11, the mass was more definite and expansile. On November 13, the wound was resutured. The varicose ulcers of the leg healed with remarkable rapidity following the operation. The wound was dressed with a solution of chlorinated soda (Dakin's solution, 1:15). A solution of full strength was applied daily with a swab to the base of the ulcers. On

November 16, the mass was smaller and not expansile. On December 26, the ulcers of the left leg were healed. On Jan. 2, 1926, the patient's general condition was excellent, however, there was a hard and fixed mass 10 by 12 cm. in the pelvis to the left of the median line. The mass could not be felt by rectum. There was no expansile pulsation. A dilated femoral artery, and probably also an iliac artery, could be felt above and below Poupart's ligament with marked pulsation. Below Poupart's ligament there was a hard mass extending into the groin. The upper part of the mass was pulsating. The appearance of the leg

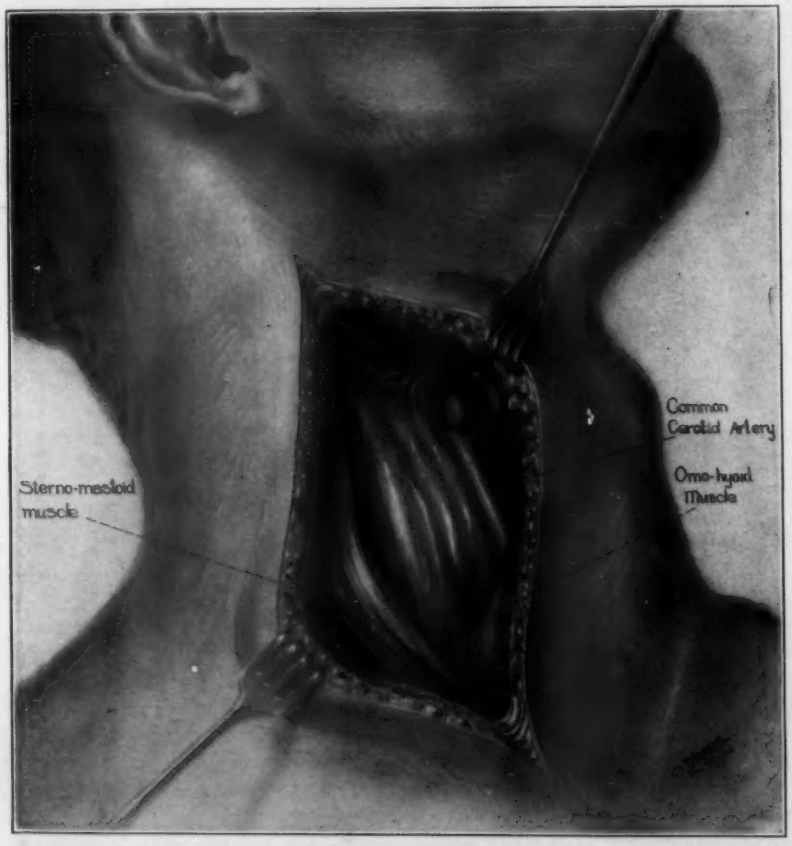

Fig. 13 (case 6).—Collapse of the internal jugular vein after ligation of the communicating branch.

was satisfactory. The mass suggested an aneurysmal condition with clotting. Exploration was advised.

Third Operation.—Operation was performed on February 8, through a low incision in the median line. There was a postperitoneal mass arising from the left pelvic wall, probably about 20 cm. in diameter. The wall of the mass was about 0.25 cm. thick. The mass did not seem to be attached to any vessel, although the left external iliac ran behind to its external surface. The first impression was that of an aneurysm from this artery, which was hugely distended, probably between 3.5 and 5 cm. in diameter and extremely tortuous. The vessel was first

isolated and a tape put around it in order to control the bleeding in case the mass should be connected with the vessel. The mass was cystic, but not expansile; it was opened, and old disintegrated blood clots were removed. Fresh bleeding did not occur. The cavity was thoroughly inspected, and there was not any evidence of an artery opening into it. Bleeding did not occur after pressure was released on the external iliac. A segment of the sac was removed, and the pathologist reported that it was lined with endothelium with some muscle in its wall. After this, the interior of the sac was reexamined with the idea of finding some communication with an artery, but this could not be demonstrated. The cavity was packed with a long double strip of iodoform gauze. It was first thought best to leave a tape around the external iliac in order to control bleeding if there should be secondary bleeding, but as the tape did not slip well, indicating difficulty in removing it later, it was decided to remove the tape. One Penrose drain was left outside of this. It would have been impossible to obliterate the sac by suture. The pathologic report was old hemorrhage and fibrin in the wall of an artery.

Fig. 14 (case 6).—Nine days after operation. Patient lived for seven years in good health without any evidence of recurrence of aneurysm, when he died following an operation elsewhere for cancer of the stomach.

For about six days after the operation, the patient's temperature was 102 F. and the pulse rate, 110; there was much cough and sputum. The gauze was shortened on the twelfth day after operation and taken out on the seventeenth day. A Penrose drain was inserted into the drainage sinus and kept in for several days. On the forty-first day, the patient was dismissed from the hospital with the wound healed; he was able to walk well with the use of a cane. On March 2, the systolic blood pressure was 102; the diastolic, 64; and on March 8, the systolic was 98, and the diastolic, 66. The result was excellent (figs. 8, 9 and 10).

This case illustrates several points of special interest. The ligature around the fistulous tract failed to close the arteriovenous communication permanently. In this instance, the tract was not definitely isolated and extravascular fibrous tissue was no doubt included in the tie, this probably accounted for the subsequent loosening of the ligature and the reestablishment of the fistula. At the time of the operation, Feb. 8, 1926,

when compression of the external iliac artery stilled the femoral artery, it was definitely proved that the pulse of the femoral artery was a continuation of the pulse of the external iliac and was not due to collateral circulation. The reestablishment of the lumen of one of the larger vessels after ligation has been noted previously. A mass formed in the pelvis. While a definite communication with the external iliac or femoral artery could not be determined, it unquestionably developed from an artery. It may have been of spontaneous origin, but I am inclined to believe that it resulted from the injury to the femoral artery in 1919, when it was found necessary to ligate it just below Poupart's ligament. The circulation of the left leg and foot was immediately improved after the destruction of the fistula. This was evidenced by the remarkably quick healing of the sloughing ulcer of the leg.

FOREIGN BODIES IN THE STOMACH

REPORT OF A CASE IN WHICH MORE THAN TWO THOUSAND FIVE HUNDRED FOREIGN BODIES WERE FOUND *

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To find foreign bodies in the stomach is by no means rare, in fact, the medical literature is so replete with reported cases that one hesitates to add another unless it is of unusual interest.

Many of the patients are children who pick up objects and swallow them accidentally or intentionally. In the normal adult the presence of foreign bodies in the stomach is usually the result of the accidental swallowing of articles held in the mouth, although at times one sees people, otherwise normal, who have the habit of swallowing hair and other objects. This habit may be accentuated in some insane patients, and it is from this group that the strangest and largest number of objects have been recovered at operation, or more often at autopsy.

REPORT OF A CASE

The following case, we feel, qualifies for a place in literature:

Mrs. H., aged 42, had been a patient in the Ontario Hospital for a year and a half on account of manic-depressive insanity. Suicidal tendencies were present, and sometimes she would pick up and swallow various foreign bodies. In May, 1926, a roentgen-ray examination (S. G. C.) revealed several of these (bent safety pins, wire and buttons) at different levels of her gastro-intestinal tract. They were not causing symptoms, and appeared to be passing through the intestine without difficulty, for a roentgenogram taken a month later showed an entirely different collection of articles. She improved somewhat mentally, became less melancholic and was not observed to be swallowing things; therefore it was thought that occupational therapy might be of further benefit, and she was allowed to go to the sewing room. Her general health improved, but at times she was seen to swallow objects. On May 20, 1927, she complained of slight abdominal pain, and a movable mass, about 4 inches (10 cm.) in diameter was palpable below and to the left of the umbilicus. On roentgen-ray examination,

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this mass was seen to consist of foreign bodies. On June 10, 1927, a gastrotomy was performed (H. O. F.) through an incision in the lower part of the left rectus. The stomach, which had been pulled down into the pelvis, was lifted through the abdominal incision, and the following objects were removed:

Objects Removed from Patient's Stomach

Bent pins	947	Washers	6
Pieces of bent wire	865	Dress hooks	5
Pieces of glass	191	Roller bearings	2
Parts of safety pins	176	Complete safety pins	2
Tacks	58	Nuts	2
Parts of corset steels	54	Broken key	1
Parts of garter fasteners	28	Sewing machine needle	1
Nails	20	Coin (American cent)	1
Buttons	17	Cuff link	1
Straight pins	9	Pen nib	1
Screws	8	Unclassified	130
Beads	8		
Total			2,533

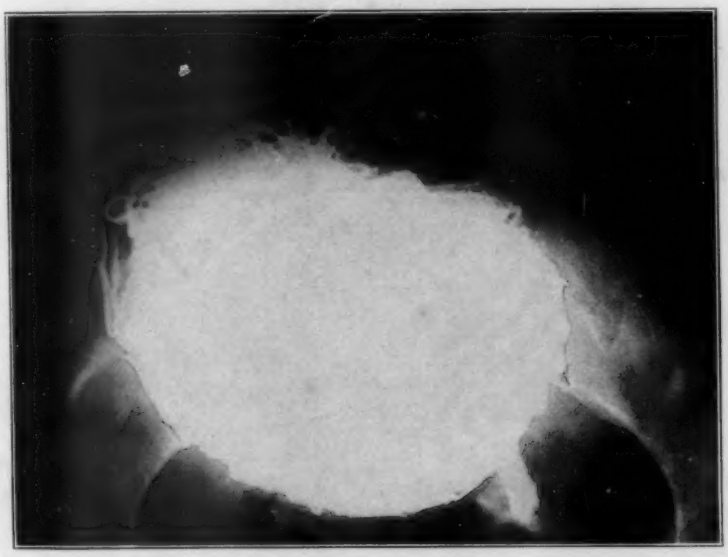

Fig. 1.—Foreign bodies in the stomach.

While most of the objects were well preserved, many of the pins, hair pins, safety pins and nails showed evidence of corrosion. The outer surface seemed harder or was protected by enamel or nickel plate, for the greatest action was on the central core, making the objects appear tubular or grooved (fig. 4 and ends of hair pins in fig. 3). It is plain that this is mainly a chemical and not a mechanical effect.

The item "pieces of bent wire" consisted chiefly of broken hair pins, while under "unclassified" were included small metallic fragments, pieces of porcelain, small stones and other objects. The aggregate weighed 410 Gm. and nearly filled a pint sealer. The majority of the articles were small, the largest being 4 cm. in length. The wall of the stomach was thickened, and contracted well after the

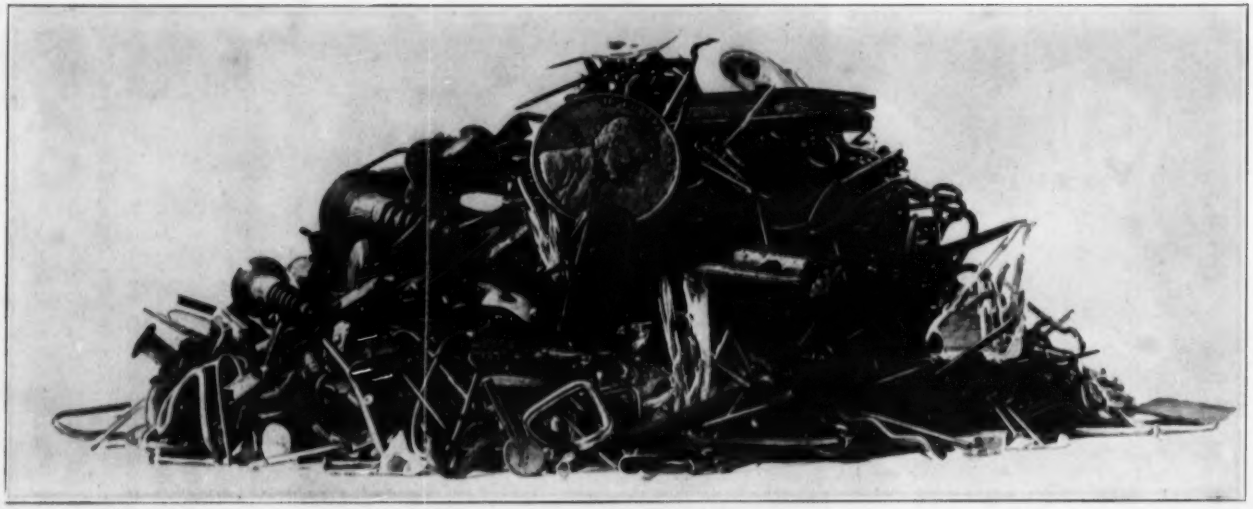

Fig. 2.—The foreign bodies piled in a heap (actual size).

load was removed, the stomach resuming its normal position. The mucosa was covered with scattered whitish plaques, as if silver nitrate had been applied. There was no gross evidence of hemorrhage, ulceration or perforation, nor were any perigastric adhesions present. With the exception of the position of the incision, there was no variation in the usual technic of the surgical procedure. The patient made an uneventful recovery from the operation.

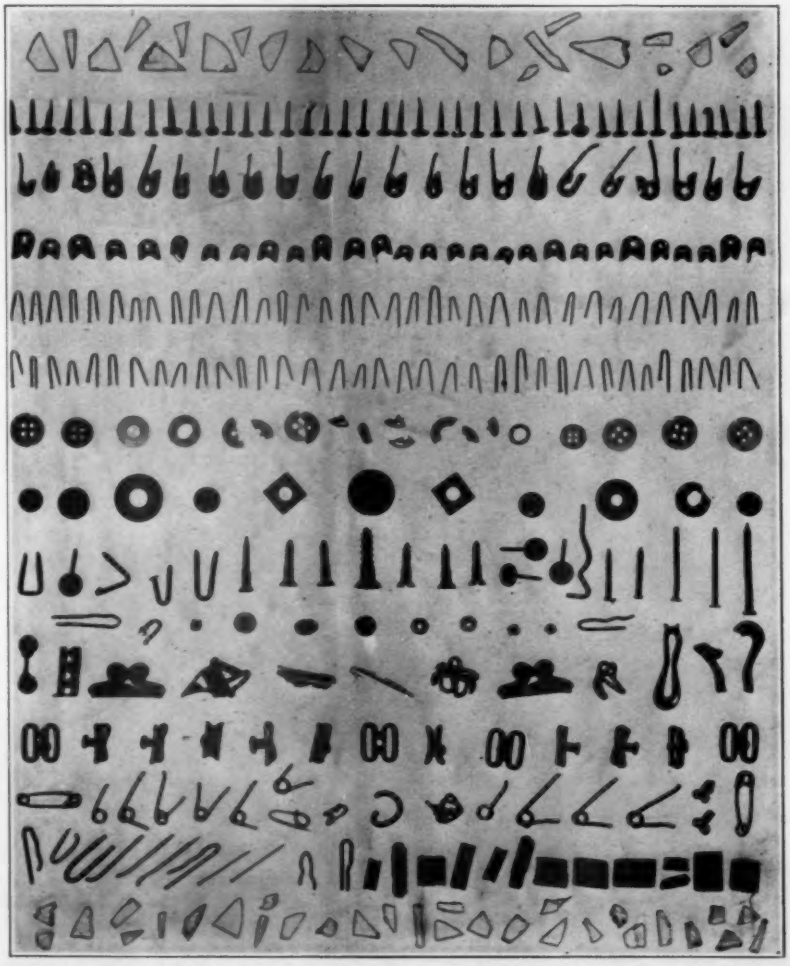

Fig. 3.—Some of the foreign bodies arranged to show greater detail (one-third actual size).

This case is of interest because of the large number and variety of objects removed at operation. The patient evidently could take care of a limited number, but over this optimum, they began to accumulate in the stomach because of the induced ptosis and the inability of the stomach to push the mass toward the pylorus and because of the interlocking of the

individual pieces. The case also illustrates the number of unsuccessful attempts at suicide. It is true that the patient took care, in most instances, to bend the pins and wire and even to bend the points of a pen nib, but this was probably to facilitate swallowing rather than to protect herself. It is remarkable that there should have been so little evidence of injury to the gastro-intestinal tract, for there were many straight, pointed

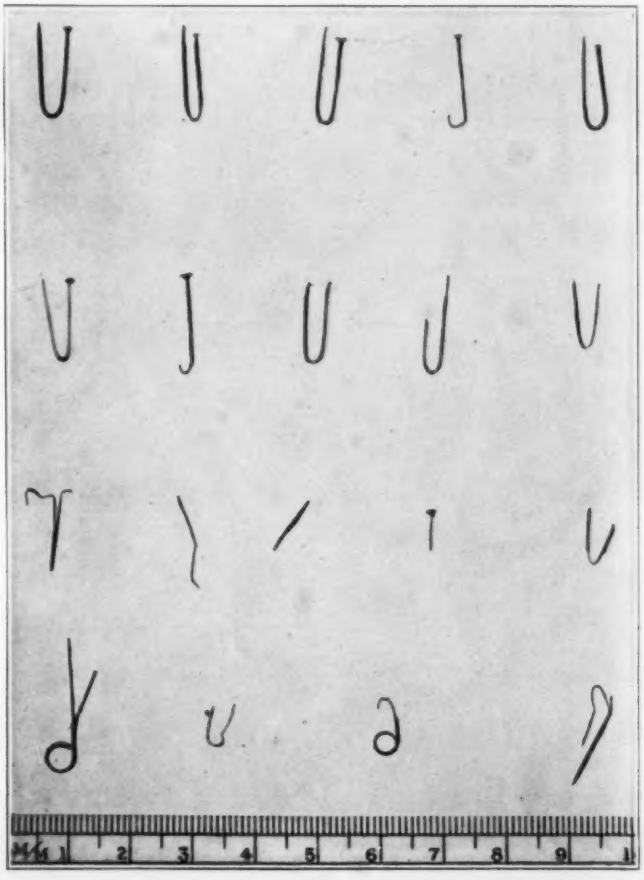

Fig. 4.—Illustrating corrosion of pins and safety pins.

and angular articles which could easily have damaged the mucous membrane. This patient is still swallowing objects. Unless she was placed in solitary confinement under close constant individual observation it would be impossible to prevent this entirely, and it would not be practical in this case. By reasonable care, however, it is hoped that the number of articles ingested may be small, so that they may have a chance to pass through the gastro-intestinal tract individually.

REVIEW OF THE LITERATURE

In 1924, Thorek,[1] reported the case of a professional swallower from whose stomach he removed 276 articles besides a quantity of bits of glass and porcelain; he reviewed the literature up to that date. Gaylord,[2] in 1903, removed 453 carpet tacks, 41 blades of small penknives, 142 screws, 40 pen-points, 6.5 ounces (184 Gm.), of ground glass and a wire chain 3 inches (7 cm.) long, the total weight being 2 pounds and 3 ounces (992 Gm.).

In 1911, Vandivert and Mills,[3] while performing an autopsy on the body of an insane woman, found, 1,446 objects—453 nails, 42 screws, 9 bolts and 942 miscellaneous articles, including buttons, safety pins, hairpins, carpet tacks and common pins—the total weight being 2,268 Gm. In this list they included 148 grape seeds and other smaller seeds, but counted a string of small beads 4 feet (121 cm.) long as a single unit. In 1913, Matthews[4] removed 1,149 hair pins, nails and other objects, while Hoisholt reported that this patient was operated on again four years later, when 921 similar objects were removed. In 1919, Winslow[5] removed from an insane patient nearly 1,300 articles, mostly small straight pins. The patient recovered from the surgical procedure. Curl and Culver,[6] in 1922, found 1,040 carpet tacks, 12 screws, 12 open safety pins and many small metallic pieces, the total weight being over 2 pounds (907 Gm.). The patient made an uneventful recovery.

In 1924, Wardell[7] palpated a mass the size of two fists in the left lumbar region of a girl, aged 16, and later removed 2¼ pounds (1,020 Gm.) of 1¼ inch (3.1 cm.) nails, which had been swallowed over a period of nine years. In 1924, Tuft[8] had a patient who swallowed two packages of straight pins (about 500) and a few safety pins, with suicidal intent. An operation was performed seven weeks later. A large perforation of the stomach with local peritonitis was found. The pins were removed, but the patient died in fifteen hours.

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1. Thorek, M.: Large Collection of Foreign Bodies in the Stomach. Report of Case with Review of Literature, Internat. Clin. **3**:282, 1924.

2. Gaylord, quoted by Friedenwald, J., and Rosenthal, L. J.: A Statistical Report of Gastrotomies for Removal of Foreign Bodies from the Stomach, New York M. J. **78**:110, 1903.

3. Vandivert, A. H., and Mills, H. P.: Foreign Material in the Stomach. Report of a Remarkable Case, J. A. M. A. **56**:180 (Jan. 21) 1911.

4. Matthews, quoted by Thorek (footnote 1).

5. Winslow, R.: Foreign Bodies in the Stomach, Ann. Surg. **70**:60, 1919.

6. Curl, H., and Culver, L. C.: More than One Thousand Carpet Tacks in the Stomach, J. Radiol. **3**:489, 1922.

7. Wardell, W. H.: Foreign Bodies in the Stomach, Canad. M. A. J. **14**: 1105, 1924.

8. Tuft, L.: Foreign Bodies in the Stomach, M. J. & Record (supp.) **119**:30, 1924.

In 1927, Blackburne [9] reported the removal of a "mass of safety pins" weighing 295 Gm. Roentgenograms were shown, but an actual count was not given.

The article of greatest weight removed by gastrotomy seems to be that reported by Swain,[10] in 1895, namely, a hairball weighing 5 pounds and 3 ounces. As far as we know, our patient enjoys the doubtful honor of having had the largest number of objects ever reported removed from any stomach either surgically or at autopsy.

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9. Blackburne, George: Mass of Safety Pins in the Stomach, J. A. M. A. **89**:1059 (Sept. 24) 1927.

10. Swain, W. P.: Case of Gastrotomy: Removal of a Mass of Hair Weighing 5 lbs. 3 oz. from the Stomach—Recovery, Lancet **1**:1581, 1895.

OBSTRUCTIVE MASSIVE ATELECTASIS OF THE LUNG*

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Pasteur described postoperative atelectasis or collapse of the lung as a condition of this organ characterized by "the total deflation (as opposed to lobular or patchy collapse) of a large area of lung tissue, of sudden onset—in the absence of any signs of obstruction of the air ways or of any known source of compression—due to failure of inspiratory power and attended by definite physical symptoms and signs."

Since then, and even long before Pasteur had again brought it up as an actuality, many papers have been written on this puzzling condition. None seems to have given a definite solution to the etiology and the mechanism of it. In this paper, we propose to give the results of our experimental work conducted on fifty-six dogs. A careful study of the clinical symptoms, the physiologic phenomena, the roentgen-ray observations on serial pictures and the pathologic and bacteriologic condition of our animals convinced us that there is but one cause in the production of collapse of the lungs, and that is the more or less temporary, but complete, occlusion of a bronchus by a plug of mucus acting as a foreign body, and intercepting completely the penetration of air into the portion of lung depending on the occluded bronchus. Complete obstruction is an indispensable factor for the production of this syndrome. The other causes concerned in its etiology act only as predisposing factors; the only actual cause being, in our opinion, the complete occlusion of a bronchus. This conclusion is based not only on our experimental observations, but on the analysis of the history of the cases published, including those presented by Pasteur.

In order to facilitate the exposition of our subject, we shall study successively in this paper the definition and value of the terms employed, the historic evolution, the etiology and mechanisms, roentgen-ray observations and the pathologic and bacteriologic processes of this condition. This paper is a general outline. A number of important points arising in the course of this work will be developed later in other papers.

DEFINITION OF TERMS

As Pasteur remarked in 1913, much confusion has arisen from the careless use of the word "collapse" in relation to conditions of the

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lung. It should only be used to indicate the condition in which "a lung or a lobe of it is completely deprived of its air contents." The expression "partial collapse" is misleading and should be replaced by "partial deflation." "Partial deflation of pulmonary bases," as it is used for bedridden patients, signifies the loss of efficient inspiratory force. It has little or nothing in common with true collapse, either clinically or anatomically, but it is doubtless predisposing to "patchy collapse" of the lung.

"Massive collapse" differs from "patchy collapse," according to Pasteur, both in its mode of origin and its clinical features. It is probably always determined by a grave failure of inspiratory power, and may be distinguished from patchy collapse in cases in which there is paralysis of respiratory muscles (diaphragm, intercostal and other muscles) by its sudden supervention without any premonitory signs of respiratory paralysis. The former variety apparently is always unilateral and includes all cases of postoperative collapse.

In order to avoid the confusion arising from the use of the word "massive collapse," Scott proposes the term active collapse or active atelectasis in order to distinguish it from passive collapse or passive atelectasis, which is due to the compression of the lung by pneumothorax, the effusion of fluid in the pleura, or even to a tumor.

Jackson and Lee prefer the term "atelectasis" to "collapse" because the latter suggests the simultaneous collapse of alveolar tissue and bronchi as it occurs in all cases of increased and positive intrapleural pressure, whereas in the so-called acute postoperative massive collapse of the lung, collapse of the bronchi does not take place. So far as "partial collapse" or "partial deflation" is concerned, Jackson and Lee consider it a common condition in the lung after death, being a lesser degree of postoperative collapse, and for that reason they do not consider it worth while to create a special terminology, thus unnecessarily increasing the already existing confusion of terms. We suggest that the term "atelectasis" always be used instead of "collapse," because, as will be seen, collapse can occur without atelectasis, but atelectasis can never occur without collapse. Even "atelectasis" is a misnomer, and "apneumatosis" should be used in its stead. The most appropriate name for the condition is "obstructive massive apneumatosis of the lung."

We believe that the definition as well as the etiology and mechanism of this condition of the lung will be much simplified if the conclusions of our experimental work are true. We firmly believe that there are only two kinds of "collapse" or atelectasis of the lung; one is due to obstruction of a bronchus, and the other to compression of the lung; the first is active, the second is passive. If we substitute for the

adjectives "active" and "passive" the etiologic attributes, we should call them "obstructive massive collapse" and "compressive massive collapse."

The definition which we propose for this condition is: obstructive massive atelectasis of the lung is a febrile complication which appears within a few days after operation, generally on the abdomen, and is always due to a more or less temporary but complete obstruction of a bronchus, and followed by the more or less complete absorption of vesicular air in the corresponding portion of lung, thus giving the organ the structure of a "fetal lung." It is especially and often conclusively characterized by the clinical symptom of a unilateral pulmonary consolidation with displacement of the mediastinum and the heart toward the affected side. Its prognosis is generally favorable and ends in lysis or crisis. The treatment is bronchoscopic aspiration of the obstructing mucus as early as possible.

HISTORY

Numerous cases are reported in the literature. It is interesting to notice that excellent descriptions and experimental studies had been made long before the relatively recent papers of Pasteur, but they were more or less completely forgotten.

It seems that the first description of this condition was given by Legendre and Bailly, French authors, in 1844, as a frequent complication of bronchitis in children. They separated it from the inflammatory consolidations of the lung, with which it was formerly confounded. They defined it as a fetal condition of the lung (état foetal), because the lung goes back to its prenatal condition. They gave as its etiology "partly the imperfect respiratory movements and partly the obstruction of the bronchi with secretions." They assigned to it a rather too simple mechanism: "As the air does not enter into a portion of the lung, the alveoli collapse because of the elasticity of the lung parenchyma."

Traube, in 1846, showed that artificial occlusion of a bronchial tube causes atelectasis of the corresponding portion of the lung, and that the same phenomena occur when the thoracic cavity is opened. But he did not believe that this was a sufficient explanation of the mechanism of the condition, and added: "We are here on the borders of a ground which we shall be able to explore only by the combination of a clinical, pathological and experimental investigation." Traube was not the first investigator to succeed in producing a "collapse" of the lung by obstructing the bronchus with a plug made with paper. In 1844, Mendelssohn obstructed bronchi with shot, paper and gum-arabic solution and produced atelectasis. He also divided the vagi and laryngeal nerves and opened the wall of the chest. The latter procedures were probably only contributing factors when atelectasis was produced.

Gairdner, in 1851, admitted that the most frequent cause of collapse is bronchitis; in this condition, the bronchi are obstructed with mucus, which acts as a ball-valve going down and blocking the funnel-like bronchus during inspiration; upward displacement during expiration allows exit of air from the bronchioles, and thus the air is gradually emptied from the obstructed portion of the lung. But this explanation, although simple, presupposes a perfect functioning of the ball-valve; even then the complete disappearance of the air in the affected portion of the lung cannot be explained. Notwithstanding the multiple objections to this theory, it met with real success at that time and Rilliet and Barthez admitted it.

Barthels, in 1860, in a great number of autopsies first demonstrated mucous secretion in the bronchi corresponding to the atelectatic portion of the lung, and later attracted attention to atelectasis of the bases, often symmetric, following bronchial diphtheria. "Diphtheria of the bronchi," he said, "is regularly followed by atelectasis of the portions of the lung corresponding to the obstructed air passages." He tried to explain the collapse by the obstacle presented to the inspiratory entrance of air into the alveoli by swelling of the mucosa and by a spastic contraction of the bronchial muscles due to inflammatory irritation of the bronchial mucosa. Air does not enter because of the resisting forces opposing its entrance, namely, the strong expiratory and cough movements. But this theory did not satisfy even Barthels, who added in his paper: "It does not seem quite satisfactory to admit that this mechanical expression of the alveolar air alone can produce collapse of lung tissue. Another factor must intervene here, especially the air absorbing capacity of the blood circulating in the capillaries encircling the alveoli, to which point Virchow first attracted attention."

The first author to study thoroughly the mechanism of massive "collapse" seems to be Jurgensen. In studying the symmetric collapse of the lowest and posterior segments of the lung, he arrived at the conclusion that it was due to incomplete respiration in those regions in which even normally the respiration is not active; therefore, in the corresponding bronchi there is an easy accumulation of mucus, which finally obstructs them. This obstruction is facilitated by the decrease of respiratory movements and cough. *Once the obstruction is established, the air contained in the alveoli is rapidly absorbed by the blood circulating in the capillaries.* This absorption is facilitated by the pressure exerted on the enclosed air by the elastic walls of the alveoli and by the surrounding lung.

Lichtheim, in 1879, published a paper which even now is the most remarkable experimental piece of work on the mechanism of "collapse." By a great number of well conducted experiments on rabbits, in which he obstructed the bronchi with sticks of laminaria, he demonstrated

beyond doubt the importance of obstruction of the bronchi in the production of "collapse," and the subsequent absorption of air by the blood circulating around them. He went further and established the speed of absorption of the different gases. Thus, operating on rabbits, he found that after obstruction of the bronchus, pure oxygen is completely absorbed in forty-five minutes and carbon dioxide in from ten to thirty minutes, whereas the nitrogen of air is completely absorbed only after twenty-four hours.

From that time till 1909, several articles were written on this question. General interest was again aroused by a series of papers published by Pasteur in England, from 1890 to 1914. This author noticed the coincidence of "collapse" of the lung with paralysis of the diaphragm, and, considering that there was a relation of cause and effect, he thought paralysis of the diaphragm was the principal factor in the production of massive atelectasis.

The description of Pasteur gave a new stimulus to the study of this question. The wars, too, presented a new opportunity for its study. Progress accomplished in bronchoscopic investigation, moreover, gave a new impetus to search out the etiology and the mechanism of this clinically well defined syndrome.

Briscoe, Scrimger, Pearson-Irvine, Schroeder and Green, Grailey and Hewitt, Rose-Bradford, Crymble, Sante and Brennemann, and especially Chevalier Jackson and his associates have published important papers on the subject. The contribution of Chevalier Jackson and his associates to the knowledge of massive atelectasis, because of the perfection of bronchoscopic investigation, has been outstanding during the last years; it is partly responsible for clearing up the etiology and treatment of this complication. Since attention has been attracted to massive atelectasis of the lung, its clinical diagnosis has become common and the condition separated from postoperative bronchopneumonia, congestion and pneumonia, with which it was previously confounded.

Despite the number of these papers, so far as we know there has not been any systematic, experimental contribution to the knowledge of the causes and mechanism of this condition since Lichtheim's remarkable work in 1879, although the use of the bronchoscope—unknown to Lichtheim—should facilitate to a great extent the experimental study and render it much more accurate. The reason for this, we believe, is the difficulty of experimentally producing, in a sure way, a "collapse" of the lung. To do this it is necessary to insist on the sine qua non condition of complete and air-tight obstruction of the bronchus in order to produce atelectasis of the corresponding occluded bronchial territory. The gradual perfection of our "occluding balloon" took much time and trial, and there were many disappointments. When the

technic was perfected, we were able to produce the condition at will, and thus study the details of mechanism, evolution, complications and the methods of treatment.

ETIOLOGY AND MECHANISM

The opinions held for the explanation of the production of collapse of the lung can be summarized as follows:

1. Compression of the lung due to paralysis of the corresponding diaphragm.
2. Inhibitory respiratory reflex.
3. Reflex spastic contraction of the bronchial muscle.
4. Obstruction of the bronchus due to angioneurotic edema of the mucosa of the bronchi.
5. Mechanical expulsion of the air through an incompletely obstructing plug of mucus acting as a ball-valve.
6. Complete obstruction of a bronchus through a plug of mucus (or a foreign body), and absorption of the alveolar air by the blood circulating in the capillaries.

Before entering into the discussion of the mechanism and etiology, we shall mention a few important and well known physiologic facts.

1. The intrapulmonary pressure is equal to one atmosphere with slight variation during inspiration and expiration, the air passages being free.

2. The intrathoracic or intrapleural pressure and pressure in the mediastinal spaces is always negative and less than an atmosphere by an amount equal to the elastic recoil of the lungs. In other words, the negative intrapleural pressure is equal to the elastic recoil of the lungs and corresponds to about 4.5 mm. of mercury at the end of expiration and 7.5 at the end of inspiration, according to the figures given by Heynsius. This was proved by Hermann, who showed that the fetal and the new-born child's pleural cavity up to the third or fourth day do not show a measurable negative pressure, owing to the fact that the elasticity of the lung has not yet come into play. It follows that although the new-born child has practically no reserve supply of air in the lung, and that at each expiration the lungs are entirely emptied, a small amount of air, the minimal air, always remains in the alveoli. But, as Howell remarks, "if for any reason the alveolar ducts or respiratory bronchioles were blocked for any length of time by mucus, for example, the air captured in the atria and air spaces might be absorbed partly or completely, giving a condition of local consolidation."

3. Each gas of the air (N_2, O_2, CO_2) in accordance with the mechanical laws of gas pressure in a mixture of gases, exerts a pressure corresponding to the proportion of that gas present. Thus, in the air, oxygen being present to the extent of 20 per cent exerts a pressure of one fifth of an atmosphere, or $\frac{1}{5} \times 760 = 152$ mm. of mercury.

4. The absorption of each of these gases in the blood, according to the law of Henry, varies directly with its pressure; that is, it increases and decreases proportionately with the rise and fall of the pressure of gas in the alveoli, the temperature remaining the same. It follows that in a mixture of gases, each gas is dissolved in proportion to the pressure that it exerts and not in proportion to the pressure of the mixture. Besides this law, we must consider the "absorption coefficient," which is the number that expresses the proportion of gas dissolved in a given volume of the liquid under one atmosphere of pressure and at a given temperature. The absorption coefficients for oxygen, carbon dioxide and nitrogen are, oxygen, 0.0262; nitrogen, 0.0130, and carbon dioxide. 0.5283 at 40 C. (Howell). The solubility of carbon dioxide, therefore, is twenty times as great as that of oxygen. Knowing the pressure in the alveoli, we may calculate how much of these three gases can be held in the arterial blood in physical solution. If we admit that in rough numbers the composition of the alveolar air is 80 per cent nitrogen, 15 per cent oxygen, and 5 per cent carbon dioxide, we find that in 100 cc. of water, the following amounts should be held in solution: nitrogen, 1.04 cc.; oxygen, 0.393 cc., and carbon dioxide, 2.64 cc. The foregoing figures are correct for water, but are not for the blood, in which, because of the presence of oxyhemoglobin, the conditions of solubility and tension for oxygen and carbon dioxide are changed. Thus, 100 cc. of blood actually contains 20 cc. of oxygen and from 40 to 45 cc. of carbon dioxide, whereas there is only 1 cc. of nitrogen which does not play a direct rôle in the physiologic processes and so is held in the blood in physical solution and absorbed by the blood in proportion to its partial pressure in the alveoli, in accordance with the law of Henry. Pflüger, analyzing the arterial blood of the dog, obtained the following figures, reckoned in per cent by volume: oxygen, 22.6; carbon dioxide, 34.3; nitrogen, 1.8.

Thus in the case of complete obstruction of a bronchus by a plug or a ligature, the intra-alveolar pressure will be increased because of the elastic pull of the alveoli, the capillary circulation of which is in contact with the contained air. This air will be absorbed by the blood, the carbon dioxide rapidly, the oxygen, less rapidly, and the nitrogen slowly. Our experiments on the mechanism of atelectasis confirm these facts.

5. The innervation of the bronchial musculature is important in a consideration of the production of atelectasis, as many authors say that the reflex contraction of the muscular ring of the bronchioli explains the shutting off of the alveoli. It is admitted that this musculature is supplied through the vagus with motor and inhibitory fibers, called bronchoconstrictors and bronchodilators. Stimulation of the constrictors

causes a narrowing of the bronchi and therefore increases the resistance to the inflow and outflow of air. Drugs known to stimulate the endings of bulbar autonomic nerve fibers, such as muscarine, pilocarpine and other preparations, seem to increase the tonus of the bronchial muscles, as well as does irritation of the nasal mucus membrane. Atropine, epinephrine, extracts of lobelia and nicotine have the contrary effect, and abolish the constriction when it exists. The last fact is important, because in a case of atelectasis, as we shall see later, atropine in high dosage was given hypodermically without causing any improvement.

These few physiologic notions will permit us more readily to attack the problems of etiology and mechanism of apneumatosis of the lung. The existing theories may be divided into three classes. The first does not admit complete mechanical obstruction of a bronchus as a necessary and indispensable condition for the production of the condition. Under this heading would come a more or less effective narrowing of the lumen of the bronchioli through spastic contraction of the muscular ring, inhibitory respiratory reflex and compression of the lungs due to paralysis of the diaphragm or the respiratory muscles. The second theory is that a mechanical valve-like obstruction allows exit but not entrance of air into the bronchus. The third theory, on the contrary, is that atelectasis is not possible without complete obstruction of a bronchus. The last is our belief and the conclusion we arrived at after long experimental study.

The exact knowledge of the mechanism of production and the etiology of massive atelectasis is not only of theoretical value. There is no doubt that "collapse," massive or patchy, constitutes one of the most frequent postoperative complications of the lung, being far more frequent than lobar pneumonia. Pasteur, Rose-Bradford, Cutler, Jackson and their associates, and almost all the modern authors agree on this point. Pasteur says: "Indeed I would go so far as to say that when physical signs, especially at the right base suggest pneumonia, the odds are in favor of massive collapse." It is admitted, furthermore, as probable that "collapse" favors the development of infections complications of the lung by microbes coming from the outside or by the fixation in the "collapsed" areas of small septic emboli coming from the operative wound, which, without collapse would not cause any noticeable trouble.

Accurate knowledge of the etiology and mechanism of atelectasis of the lung, therefore, is of great interest to the surgeon, because it would enable him to prevent the occurrence of this condition or to treat the patient efficiently.

The theory advanced by Pasteur, i. e., that massive "collapse" is due to paralysis of the diaphragm, in our opinion, cannot be admitted today as a sufficient cause. Pasteur reports sixty-four cases of postdiphtheritic

paralysis, in twenty-eight of which the diaphragm was involved; fifteen of these had a fatal issue, and autopsy was performed in eight. In five of these, there was clear evidence of paralysis of the diaphragm for several days before death, and in each of these cases the lower lobe of the right lung was collapsed and entirely devoid of air. This coincidence is impressive, but there were two cases of "collapse" in which paralysis of the diaphragm was not present, and a third in which the paralysis had disappeared twenty-four hours before death. The absence of paralysis of the diaphragm in those three cases greatly weakens Pasteur's theory. There are other far stronger arguments against this theory, especially physiologic ones.

The mechanism suggested by Pasteur is as follows:

"As the expression of the air of the lungs at birth is primarily due to muscular effort and is subsequently maintained by the balance between the forces which cause expansion and contraction, respectively, it follows that whenever—whether as the result of paralysis or of temporary inhibition—the distending forces acting on any part of the lung becomes less than that of the agencies which tend to cause it to contract, the latter forces take charge, with the result that the part of the lung concerned rapidly empties itself of air." Pasteur claims that the mechanism here suggested—active contraction—differs from that generally accepted in explanation of collapse, namely, gradual absorption of the contained air from a portion of the lung which has been shut off from communication with the external air by occlusion of the air ways. He accepts only two causes in the production of massive "collapse": compression of the lung by the diaphragm and diminished respiration.

But this cannot explain the complete emptying of the air in the lung by the slight compression due to the ascension of the paralyzed diaphragm. The minimal air remains in the lung even after wide opening of the thoracic cavity, and only later is the air gradually absorbed, if the animal survives. Even with marked compression of the lung by abundant effusion of fluid or pneumothorax, the absorption of the minimal pulmonary air is gradual, the carbon dioxide being absorbed most rapidly, then the oxygen and finally the nitrogen; this shows that the disappearance of the air is due to a more complex mechanism than a merely mechanical one. The same phenomenon occurs in the ligature of a bronchus (Lichtheim). In these cases, the absorption of air obeys the same physicochemical laws.

The often reported sudden onset and disappearance of the symptoms of atelectasis after expectoration of thick sputum cannot be explained by this theory, although Pasteur believes that the bronchus of the affected lobe is secondarily filled with secretions. It is unfortunate that Pasteur does not indicate in the reports of the cases which came to autopsy whether or not mucous plugs were present in the bronchi.

This is mentioned in only two cases: in case 10, in which the bronchi were empty, and in case 2, in which there existed "a general dilatation of the bronchi which exuded pus on pressure." He also quotes a case of Samuel West in which there was "collapse" following plastic bronchitis. The report made at autopsy reads: "An extensive collapse of the left lung developed consequent to obstruction of the main bronchus by a cast."

Paralysis of the diaphragm alone does not seem to produce great disturbance in the respiratory mechanism. The great majority of authors have not noted atelectasis of the same lung when phrenicotomy has been performed for tuberculosis (Sauerbruch) or in experimental sections of one or both diaphragmatic nerves in the neck in animals. Lemon, in his recent paper on the physiologic effects of phrenic neurectomy, stated that he sectioned one or both phrenic nerves and did not find atelectasis of the lungs either during life or at autopsy. He noticed congestion, but not atelectasis, only in a dog on which he performed double phrenicotomy. "In examination of animals," he said, "several weeks after phrenic neurectomy it was impossible to determine with exactness the side that had been operated upon when only physical examination and tambour readings were available." In the roentgen-ray examination, the heart of this dog was found in its normal position, and alteration of the normal density of the lung could not be observed. Changes did not occur in the intrapleural pressure on the operated side or in the functional respiratory capacity of the animals before and after phrenicotomy. Sections of the lung did not show any change. So far as the movements of the thoracic wall are concerned, Lemon said that they are not influenced by phrenicotomy in either extent or direction.

In order to check up Pasteur's theory, Elliot and Dingley performed phrenicotomies on cats and rabbits. They did not notice any pulmonary changes. In other cats they performed hemisection of the spinal cord at the second and third cervical segments, respectively; complete paralysis of the intercostal muscles and the diaphragm of one side followed. After three days, the animals were killed; "collapse" was not found in any case.

Crymble reported a case (case 15) in which a complete paralysis of the diaphragm and respiratory muscles of the same side occurred as a consequence of a traumatism caused by a shrapnel ball at the height of the fifth cervical vertebra (direction of the shrapnel from the fifth to the third cervical vertebra). Atelectasis did not occur. A great number of phrenicotomies have been performed for tuberculosis during recent years. So far as we know, atelectasis of the lung has not been reported to date. We find in the literature, however, a number of cases in which paralysis of the diaphragm was followed by massive atelectasis.

Briscoe reported various degrees of "collapse" in rabbits after phrenicotomy, but, curiously enough, the collapse was not limited to the affected side, but was often bilateral and frequently was more extensive on the other side. We believe that in these experiments there were probably congestive lesions of the lungs. Martin and Hare are quoted by Pasteur as having noticed "bilateral collapse" in an animal dying after a bilateral phrenicotomy, and Schroeder and Green admitted that phrenicotomy could produce "collapse" of the lower lobe. The contentions of the last authors do not seem to be backed by sufficient experimental proof.

Rose-Bradford, in his exhaustive paper, did not admit the theories of Pasteur. He analyzed cases of "collapse" secondary to gunshot wounds. In the cases of perforation with hemothorax, he explained the production of "collapse" by the possible blocking of a bronchus by clotted blood or by compression of the lung. The explanation becomes more difficult in the cases in which there was a slight wound of the thoracic wall without penetration or even fracture of a rib. In these cases, the "collapse" may not only be homolateral, but even contralateral. "Collapse" constitutes a rather frequent complication of a slight thoracic wound, about 10 per cent, according to Bradford. One of the most typical cases reported in his paper is that of a soldier who received a superficial wound in the wall of the chest; he walked four miles to a dressing station, where the bullet dropped out of the wound. He was sent back to the base hospital, and did not complain of any symptoms. The same night he was found to have a contralateral "collapse." Bradford explained these cases in particular and the collapse of a lobe or more by "immobility and retraction of the chest wall due to constrained posture and prolonged recumbency; insufficient expansion of the chest whenever prolonged is capable of causing collapse." He further added: "If as a result of cessation of respiratory movements the chest should become fixed in an extreme expiratory phase, the air in the lungs would not be adequately removed and the air present would be absorbed by the blood-stream; thus collapse might be brought about."

This theory is contrary to physiologic facts, for it is inadmissible that complete evacuation of a pulmonary airway would be produced by expiration, however extreme, with absorption of the air by the circulating blood under these conditions. Although Bradford said that "the production of this chest-wall condition remains most obscure," he remarked on the other hand, that "there is definite post-mortem evidence that massive collapse may be present and yet no obstruction of the bronchi be found. Hence, obstruction of the main bronchi may be excluded as a common or probable cause of the condition." Yet he admitted that obstruction of small bronchioles may be sufficient cause

of patchy "collapse." We quote the opinions of Bradford extensively because they have gained considerable recognition in most papers written concerning massive "collapse."

In opposition to the opinions expressed by Thoenes, Wallgren and other authors that the condition is due to compression by emphysema of the other lung, Rose-Bradford does not concede the existence of such a condition of the other lung, although he admits it in patchy "collapse" complicating bronchitis. He does not believe that spastic contraction of the bronchial muscles can produce atelectasis, because "never in asthma cases, in which this condition exists, does massive collapse occur."

It is evident that the theory of paralysis of the diaphragm and of other respiratory muscles cannot alone explain the production of atelectasis. We have repeated the experiments of Lemon on dogs, and our results agree with his. We have never been able to produce atelectasis by unilateral or bilateral phrenicotomy. Jackson, Lee, Scott, Tucker and others showed that in diphtheria the obstruction of bronchi by membranes often produces atelectasis. Systematic and routine aspiration of these membranes by bronchoscopy gave Jackson and his associates remarkable results in the ward for patients with diphtheria in the Municipal Hospital in Philadelphia. "This post-diphtheritic collapse, described by Pasteur," said Jackson, "is really due to bronchial obstruction of the air passages by diphtheria exudates and by the routine removal of these exudates we have been able to prevent it."

In our opinion, the sequence of phenomena would be: obstruction of a bronchus, absorption of the alveolar air, shrinkage and decrease of the volume of the lobe supplied by the obstructed bronchus. As a consequence of the negative intrapleural pressure, there is flattening of the wall of the chest, overdistention of the other lung, displacement of the heart and mediastinum toward the affected side and elevation of the diaphragm, which is "sucked" into the thoracic cavity of the same side. Thus the ascension of the diaphragm would not be the cause but the result of atelectasis. Even in cases of undoubted paralysis of the diaphragm this is only an indirect cause, and by the slowing-down of respiration in the lower lobes, it favors the accumulation of secretions in the corresponding bronchi. Other indirect causes are limitation of thoracic movements, prolonged recumbency and diminished cough ("the watch-dog of the lungs," as Jackson calls it). Cough is inhibited because of pain from traumatisms (even superficial) of the thoracic wall, operations on the abdomen (more especially those on the upper part) and lesions of the abdominal cavity. This theory explains the rarity of atelectasis in lesions, operations or wounds of the lower extremities and its total absence in operations on the head or upper limbs. This explanation not only sounds simple and logical, but has its basis in "col-

lapse" observed in obstruction of bronchi by foreign bodies and in experimental obstruction. The usual agent of this obstruction appears to be a plug of mucus. Before studying the arguments in favor of obstruction of a bronchus, we deem it necessary to examine rapidly some other theories proposed to explain massive "collapse."

Under this heading, we find: incomplete mechanical obstruction by (1) a reflex contraction of the bronchial muscles, (2) a swelling of the mucosa (Briscoe) comparable to angioneurotic edema (Scott) and (3) a vasomotor reflex (Scott). These authors bring in clinical and experimental support in favor of the reflex idea as a determining cause of massive "collapse." The clinical symptoms are the sudden onset, the persistence of tubular breathing over the affected area and the rapid change in stethoscopic symptoms as in case 38 of Scott, in which musical râles were present in abundance throughout both sides of the chest when the patient was examined. When the patient was reexamined half an hour later, only a few râles were present on the affected side and some on the opposite side; furthermore, the vital capacity showed a greater diminution than would be expected from complete suppression of the function of a lung. Experimental data in favor of the reflex mechanism are those obtained during the experimental work of Carlson and Luckhardt, who produced contraction of the "sack-like" lung of amphibians by electrical or mechanical stimulation of the nasal passages, small and large bowel, kidney and bladder; also the bronchial spasm or swelling of the mucosa in anaphylactic shock in the guinea-pig.

While the foregoing reflex phenomena may be contributory causes by facilitating the accumulation of mucous secretions in the bronchial tree with subsequent occlusion, we believe that they alone are unable to produce atelectasis. *Spastic contraction of the bronchial muscles, moreover, produces emphysema and not "collapse";* likewise emphysema is produced in (*a*) reflex asthma due to irritation of the nasal membrane; (*b*) allergy or (*c*) anaphylactic organ-shock in the lung of the guinea-pig. The contraction of the bronchial muscles, which was considered a cause of atelectasis of the lung, certainly occurs in acute anaphylactic shock in the guinea-pig. The dominant pathologic feature in these animals is the tetanic contraction of the smooth musculature of the bronchioles (Auer and Lewis), owing to hypersensitiveness of the smooth muscle tissue, which is also observed in other organs in the same animal. The lung in these animals is not only not "collapsed," but, on the contrary, is inflated. This inflation not only remains maximal after the removal of the lungs from the chest, but resists reduction with moderate pressure (Coca).

The lung in the rabbit is rather diminished in volume, owing to the spactic contraction of the arterioles of the pulmonary artery, but the

bronchial tree is free and "if artificial respiration is carried out the inflation and deflation of the lungs occur with not the least hindrance" (Coca). As would be expected, atelectasis of the lung has never been found in this animal.

The objection might be raised that the guinea-pig dies so quickly after the lethal injection that atelectasis cannot develop. If so, the same objection should weaken the validity of the reflex theory applied in cases of sudden atelectasis in man, concerning which precisely the same theory—bronchial spasm or angioneurotic edema—has been proposed. There is a stronger argument in what happens in protracted anaphylactic shock in the guinea-pig. As Auer stated, even in sublethal shock the lungs are always at least partly inflated. Hoover suggested that the inflation of the lung in the guinea-pig is due to edema of the mucosa and not to tetanic contraction of the bronchial muscles. One may well wonder if Hoover is correct about the merit of the explanation that atelectasis in man is the result of "angioneurotic edema"; this theory is upheld by Bergamini and Shepard to account for their two rapidly fatal cases. Swelling of the mucosa without the presence of a plug of some kind is not sufficient to cause the condition, because, as we shall try to show later, a complete and absolute obstruction of the bronchus is indispensable for the production of massive atelectasis. For the same reason, we cannot agree with the suggestion of Sante that "most probably some infection or insult to the region of the vagus supplied produces a reflex action on the bronchi permitting their temporary collapse; once approximated the walls of the bronchioles are held in opposition by cohesion, collapse of the lung rapidly following due to absorption of alveolar air by the circulating blood." Whatever the origin of the stimulus may be, the reflex must be transmitted through the fibers contained in the sympathetic and pneumogastric nerves and their respective centers. We have already mentioned that atropine given subcutaneously abolishes the action of the bronchoconstrictors. In the case of Scott, 2.75 mg. were given within thirty minutes in three doses, while the patient was under fluoroscopic examination. Modification in the condition of the lung or displacement of the heart was not noticed. This fact shows clearly that when the atelectasis has set in, it is no longer under the influence of the reflex that may have induced it by favoring accumulation of mucus in a bronchus which finally became completely obstructed. Our own experimental work clearly demonstrated to us that massive atelectasis cannot occur if the obstruction is not absolutely complete, or it will cease to exist when the obstruction ceases to be complete.

It seems that in the present state of our knowledge concerning the etiology and mechanism of massive atelectasis, we must distinguish the

contributing causes from the determining cause. The first are (1) embarrassment of respiratory movements of the wall of the chest or of the diaphragm from any cause, be it paralytic, reflex, as from pain, or mechanical, as from posture; (2) increased bronchial secretions caused by irritation or infection of the bronchial mucosa, bronchitis or pneumonia; (3) decrease or abolition of the cough reflex ("the watch-dog of the lungs," as Jackson picturesquely calls it) as a means of avoiding pain or due to the use of narcotics. There is only one determining cause, i. e., the complete obstruction of a bronchus by a plug acting as a foreign body, or by compression from the outside.

The theory of the obstructive nature of atelectasis is not new. As we have already mentioned, Mendelssohn, in 1845, and Traube, in 1846, succeeded in producing this condition by plugging the bronchus with paper balls. Lichtheim, in 1879, published the results of his remarkable experimental work in which he used rabbits, plugging their bronchi with sticks of laminaria introduced through an opening in the trachea and controlling the results by opening the thoracic cavity from time to time. Although he could not use either the bronchoscope or the roentgen ray, he obtained accurate results. He was not only able to produce a typical atelectasis each time, but he showed that the carbon dioxide was absorbed far more quickly than the oxygen, and the oxygen more quickly than the nitrogen, in accordance with physico-chemical laws. This was the first demonstration that atelectasis is due to absorption of alveolar air by the blood circulating in the alveolar capillaries. Plugging of a bronchus or ligature of it without ligation of the pulmonary and bronchial vessels was followed by complete absorption of the contained air within twenty-four hours. But if the vessels are comprised in the ligature, the lung becomes edematous and more or less necrotic, and the air does not completely disappear. The paradoxic occurrence of the edema of the lung (which means persistence of circulation to some degree even after ligature of the pulmonary artery, the vein and even the bronchial vessels) is due to the fact demonstrated by Küttner that small branches are given to the lung from the pericardial, esophageal and mediastinal vessels.

According to Lichtheim, after ligature or complete obstruction of a bronchus, the corresponding lobe or lung in rabbits becomes airless in from about two and one-half to four hours. During the first hour, the decrease of volume is rapid, but the lung conserves its normal color. During the second hour, the lung shrinks more and more, and on its surface black-red islands appear which gradually become confluent as the lung becomes airless. This irregular appearance of atelectasis is due to the fact that the activity of the capillaries of the lung, as well as of other organs, varies in different parts of the

parenchyma; consequently, the absorption of air does not take place simultaneously in the whole parenchyma of the portion that is becoming atelectatic.

Andrews, in his experiments on the cardiorespiratory function following atelectasis of one lung by bronchial ligature, as well as Scrimger (quoted by Archibald in the discussion which followed the reading of Andrews' paper), found that after ligature of a bronchus the lung becomes definitely atelectatic within from twenty-four hours to three days after the operation, owing to complete absorption of air.

Jackson, Lee, Tucker and others contributed greatly to the demonstration of the relation between obstruction of a bronchus and atelectasis of the corresponding portion of the lung. They have demonstrated repeatedly that atelectasis of the lung follows obstruction of the bronchus, and that the liberation of the obstructing foreign body from the bronchus is always followed by disappearance of atelectasis. Furthermore, this phenomenon is so common that in the bronchoscopic clinic of Chevalier Jackson, it is used by the radiologist, Dr. Manges "as a diagnostic sign of nonopaque foreign bodies or of plugs of secretion that are essentially foreign bodies." That the relation between the obstruction of a bronchus and atelectasis is clear is proved by the following quotation of Jackson's: "In a number of cases the foreign body has shifted, causing atelectasis in different lobes not simultaneously but in succession. It has been very interesting to have Dr. Manges tell us in a case of shifting foreign body, 'now the prune stone is causing obstructive atelectasis of the lower lobe'; the next day, 'now it is causing obstructive atelectasis of the upper lobe'; later, 'now it is causing obstructive emphysema of the upper lobe with obstructive atelectasis of the middle lobe.'"

But there is a sine qua non condition for the production of atelectasis, and this is that the obstruction must be complete. In partial obstruction allowing ingress and exit of air ("pass valve" idea of Jackson), nothing will happen. If ingress is allowed but exit checked ("check valve"), an emphysematous condition will occur; complete absorption of the air takes place if both ingress and exit are stopped ("stop valve"). The lung will present the structure and appearance of a fetal lung.

But can this absorption of the alveolar air by the circulating blood be demonstrated experimentally? Furthermore, is it possible to give experimental proof that it is the only explanation? In other words, is it demonstrable that there can be no other mechanism for the production of atelectasis than complete obstruction (temporary or not) of an airway of any size corresponding to the area of the affected lung?

The solution of the questions has been the aim of our experimental work. Before giving our results, we shall state what has been known about the subject from the time of Lichtheim's paper in 1879 (from

which latter we shall borrow liberally) up to the present day. It is known that atelectasis may occur following (1) ligature of a bronchus, (2) obstruction of a bronchus, (3) opening of the thoracic cavity and (4) compression of the lung by fluid or air present in the thoracic cavity. The question arises whether the real cause of atelectasis is always the absorption of the air by the circulating blood.

1. *Atelectasis Following Ligature of a Bronchus.*—We have seen that carbon dioxide, oxygen and nitrogen of the air are completely absorbed in a time varying according to the size of the animal, from two and one-half to four hours in rabbits and three days in dogs, and that the carbon dioxide is absorbed more quickly than the oxygen, and this more quickly than the nitrogen. These are experimental facts. There are now fewer difficulties in the way of explanation of absorption. In fact, absorption of alveolar air by the blood of the capillaries can take place only if the absolute and relative tension of the gases of the alveolar air is greater than the tension of the same gases in the blood. This is possible only if it is admitted that the elasticity of the tissue of the lung is not exhausted before a complete contact of the alveolar walls is effected. If the alveoli of the lung were like myriads of toy balloons in which, even after complete spontaneous deflation, a cavity with air still remained, an equilibrium would be established between the gases of the alveolar air and of the circulating blood, and no more absorption would occur.

Against this hypothesis of perfect elasticity of the lung is, in appearance at least, the fact that if the thoracic cavity is opened widely (without ligation of the bronchus), the lung collapses but does not become atelectatic, i. e., airless. The minimal air still remains, and a piece of this lung floats in water and gives off air bubbles when expressed under water. The same aeration is observed in the lung of the cadaver. This objection, however, is only apparent, because in the living animal (as Traube first showed), the lung becomes blackish blue and airless in from one to two hours after the opening of the thorax. The explanation of the deferred atelectasis is that because of collapse of the walls of the small bronchioles, of the contraction of their muscular ring (Bartels) and of the length of the bronchial tree, a resistance to the complete exit of air is offered; but the air which remains in the alveoli is compressed above atmospheric pressure, and so is absorbed by the circulating blood. This is obvious, because if the alveolar air were not under a pressure greater than the atmospheric air, if the bronchi were open, air would enter the alveoli to replace that which had been absorbed, if absorption could take place under such conditions. This consideration leaves no doubt that the elasticity of the lung is not exhausted until all the air is expelled (or rather, absorbed) and until the lung comes back to its prenatal, atelectatic condition.

2. *Atelectasis Following the Obstruction of a Bronchus, with a Closed Thoracic Cavity.*—The principles of absorption remain the same; but the explanation which may be true for the open thorax may not apply to the closed thorax. During the absorption of the air, the volume of the lung diminishes; because of the negative intrapleural pressure, the other lung expands gradually, and the mediastinum and the heart are displaced in order to fill up what may be pictured as the emptied space. In reality, there can be no such space. The contractility of the elastic tissue of the obstructed lung, however, will be opposed by (1) the stress and greater tendency to elastic recoil of the other (overdistended) lung and (2) the resultant pull on the mediastinal structures; the more the obstructed lung shrinks, the greater will these last two forces tend to become. What is the strength of these forces opposing collapse—forces which may diminish intra-alveolar pressure of gases and thus hinder their absorption? In order to calculate theoretically the alveolar pressure in the obstructed lung at a given moment, the value of the elastic overdistention of the other lung must be subtracted from the atmospheric pressure. Inversely, if even under these conditions total absorption of the alveolar air should occur, we should have to conclude that the elastic recoil of the obstructed lung is strong enough to counteract the resistance of the aforementioned opposing forces. Practically the pressure necessary to hyperdistend a lung is small and almost negligible, being equal to a fraction of a millimeter of mercury. The theoretical forces tending to lessen intra-alveolar pressure in the obstructed lung are thus also negligible and obviously cannot modify the intrapleural negative pressure.

The foregoing considerations hold equally well for patchy "collapse," which so frequently complicates bronchitis, and which is so often observed at autopsy, especially when the patient has undergone an abdominal operation. The only difference between patchy and massive atelectasis is in the size of the bronchus obstructed by a plug of mucus. Experimental as well as clinical observations leave no doubt about this point.

3. *Atelectasis with the Airways Free but with the Pleural Cavity Wide Open.*—It is understood that this experimental work cannot be carried on, without intratracheal insufflation in dogs, in which complete permeability of the mediastinum would not allow the animal to survive even after only one pleural cavity was opened. Monkeys, calves or rabbits are more suitable animals for this study. We have said that even in these animals the lung becomes completely airless after a short time (from one to two hours in rabbits) when the chest remains open. Is this due to absorption by the circulating blood? We think there can be no doubt that it is. The proof is given by what happens after ligation of the pulmonary artery. As Lichtheim first showed, several

hours may elapse between the ligature of the pulmonary vessel and the establishment of a hemorrhagic infarct. Thus, since the normal lung (with the chest open) becomes completely "airless" in from one to two hours, there is a sufficient margin of time for comparative observation and deduction. From six to eight hours after the ligature of the pulmonary artery, the lung will become smaller because it has been emptied of blood; but only its surface, to a depth not exceeding from 1 to 2 mm., has become atelectatic. Its center is full of air. This airlessness on the surface is a phenomenon independent of the circulation, because it is observed even in the cadaver's lung when it is exposed to the air, and is due to the expression of the alveolar air through the alveolar walls. That the air is not expressed through the airways is proved because: (1) the phenomenon is not observed in the cadaver when the thoracic cavity is closed (when there is no air around the lung), and (2) the same phenomenon is noticed even when the lung is extracted from the chest and one of the main bronchi is ligated. In the second instance, both lungs contain the same amount of minimal air at the moment; the peripheral atelectasis will advance to the same degree in both. According to the foregoing facts, there can be no doubt that the principal cause and the mechanism of atelectasis is the absorption of alveolar air by the blood.

4. *Atelectasis Caused by Compression of the Lung by an Exudate.*—The mechanism, in our opinion, remains the same. The portion of the lung submerged in the fluid is in the same condition as if the thoracic cavity were open. The intrathoracic negative pressure is absent, and thus the part of the lung submerged collapses exactly as if the chest were open; the remnants of minimal air are gradually absorbed by the blood. The proof of circulatory absorption lies in the fact that atelectasis is noticed not only when there is abundant exudate filling up practically the whole pleural cavity, but even when there is a moderate amount. The process is to a great extent independent of the quantity of fluid. Yet even in this condition the possibility of bronchial obstruction by an accumulation of mucus due to disturbance of respiratory movements must not be forgotten.

We have told what we know about this puzzling question of atelectasis of the lung. We have tried to give as complete an exposition of the mechanism and etiology as we were able. Many phases of the question seem to have been cleared up during the last few years, especially since Pasteur called the clinician's attention to the condition and the possibility of its clinical diagnosis. The roentgen-ray and bronchoscopic examinations have helped in no small measure. Yet, is it not true that even the most recent papers on this question show that there is great uncertainty in the minds of the authors?

We thought it would be worth while to try to clear up this question by experimental work conducted with the aid of the bronchoscope and roentgen-ray control. Since the work of Lichtheim, we know of no systematic experimental research that has been made concerning this subject, despite the fact that he published his paper in 1879, long before the roentgen ray and bronchoscopy were known!

The problems we investigated are:

1. Phrenicotomy unilateral and bilateral; study of the respiratory rhythm and the intrapleural pressure; obstruction of bronchi in phrenicotomized animals on the same and on the opposite side of the paralyzed diaphragm.

2. Obstruction of a bronchus and investigation of atelectasis under direct observation, the animal being in a box with oscillating negative pressure.

3. Obstruction of a bronchus with our obstructing elastic balloon and observation of the evolution of the "collapse" by fluoroscopy and serial roentgenograms. Autopsies have been performed and sections have been made on all our animals.

GENERAL TECHNIC

Operations were performed under general anesthesia, which was administered by intraperitoneal injection of alkaline solution of iso-amyl-ethyl barbituric acid 10 per cent (10 Gm. dissolved in 88.5 cc. of half normal sodium hydroxide carbonate-free). This gave us an excellent anesthesia, the effects of which lasted from four to nine hours. The iso-amyl-ethyl barbituric acid does not influence the cardiorespiratory system, although large doses slow down the number of respirations. A few times we used ether in intratracheal anesthesia with the Connell apparatus.

The phrenicotomies were performed under strict asepsis. The phrenic nerve was found by an incision on the posterior border of the sternomastoid muscle, and avulsion was performed after section of every root or branch which seemed to have any connection with it. In this operation we followed the method described by Lemon. In making respiratory graphs on these animals, we used Lemon's method, slightly modified. Instead of using the stand constructed by this author and having the two pneumographs which were applied respectively to each part of the thorax of the animal and posteriorly and laterally on the frame of the stand, we fixed them directly on the animal. For that purpose, two small hooks were screwed posteriorly on the spinal process of the seventh or eighth thoracic vertebra and anteriorly on the middle of the sternum. Sometimes the pneumographs were fixed by a thread, instead of by hooks, to the integument posteriorly and anteriorly. This is a poorer procedure because of the mobility of the skin. We constructed special pneumographs, using sensitive coils of fine brass wire covered by fine rubber tissue. When connected with the tambours, the airtightness was checked up under water. The reason for the foregoing modification is that without it the animal can move independently of the pneumographs during the experiment. Even if this movement is slight, the change of position of the pneumographs on the wall of the chest is followed by changes in tension of the

coils and consequently by changes in the amplitude of the graphs. We avoided this to a great extent by fixing the instrument directly on the animal.

The intrapleural pressure was measured by connecting a double water manometer comparable to the one used by Lemon or two tambours for tracings with two thoracic cannulas, as used in our physiology department. The cannulas were thrust into the respective thoracic cavity through the intercostal space after a small incision had been made in the skin. In this way we avoided any leakage between the cannula and the wall of the chest and any injury of the delicate tissue of the lung of the dog which would produce pneumothorax.

For the obstruction of the bronchi we used a specially constructed inflatable balloon introduced through the Jackson bronchoscope. The construction (fig. 7) of this balloon has been the most laborious and difficult part of our technic. We were looking for a device which would obstruct a given bronchus completely or incompletely as we wished, and the degree of inflation of which we would at the same time be able to check up with the roentgen ray. In this way we would be able to determine the relation between the degree of obstruction and the degree of atelectasis produced. Previous attempts at obstruction with foreign bodies of various kinds, such as metal, cork, laminaria sticks and plugs of cotton, had been unsuccessful. In its final form, our obstructing balloon is composed of a small glass tube, 4 cm. in length and a little less than 0.5 cm. across, the proximal opening of which is narrowed in the flame so as to be almost punctiform. A rectangular piece of fine rubber tissue (old glove) under slight tension is tied over the distal opening, forming a one-way valve. The tube and valve are introduced into a piece of thin rubber tubing, closed at one end, a little more than 0.5 cm. wide and 4 cm. long. The best rubber tubing for this purpose is that used on the Kollman urethral dilator. An outer covering of fine organdie is passed over this tube, which is wide enough to permit the system, when inflated, to have a diameter of 1.5 cm. or more. The rubber tube and its covering are twisted so as to express the air and diminish their volume and are then tied up near the proximal end of the glass tube, leaving about 1.5 cm. of the latter free. Superfluous rubber and material are cut away near the neck of the glass tubing. In this form, however, the balloon can easily be expelled by coughing, and in our experiments this happened repeatedly. Once it obstructed the larynx causing the death of the animal by asphyxia. For that reason, we fixed on its proximal end a loop of steel piano wire to catch the walls of the bronchus and oppose any tendency to displacement. The extraction of the balloon with the bronchoscope is easily accomplished by a long forceps grasping the wire. The balloon is thus completed. Air or water can be introduced into the balloon but cannot escape; they can remain well inflated for a long time, although sometimes they gradually become disinflated. This slow, spontaneous disinflation has been useful in our work. The balloon is connected by a piece of fine rubber tubing (not over 2 cm.) to a glass tubing 45 cm. long and of the same diameter as is used for the glass tubing of the balloon. This leaves 5 cm. of tubing protruding from the bronchoscope when the balloon is in place. A 10 cc. syringe filled with concentrated potassium bromide solution is connected with the long glass tubing at its proximal end. Before connecting the balloon to the distal end of the long glass tubing, the latter is filled with the same solution in order to expel the air. The whole system is now ready for use.

The Jackson bronchoscope (9 mm. in diameter) is introduced, the same technic being followed as in man. When dogs are used the only difference is that because of the straightness of the cervical vertebral column and the dimensions of the mouth of the animal, the introduction is much easier and a laryngoscope

is not necessary. In order to keep the mouth of the animal open, we constructed a special gag, using a rectangle of wood reinforced on the two sides with lead plate. The carina and then the main and secondary bronchi are located with the bronchoscope. When this has been done and the bronchoscope has been well fixed, the long glass tubing with the balloon attached is introduced. As the exact length of the system is known, one can easily calculate how far the balloon is from the tip of the bronchoscope; after a little experience, one can easily tell when the wire of the balloon has passed through the tip of the bronchoscope. The balloon is now inflated by injecting potassium bromide solution into it from the attached syringe. The amount varies greatly with the nature and size of the animal and with the form and size of the balloon. Generally from 5 to 7 cc. are necessary for a balloon such as the one described and for a dog that weighs from 9 to 10 Kg. The syringe is now disconnected and a nonflexible iron rod (an ordinary piece of hay wire is suitable), 50 cm. long and about 1 mm. wide, is introduced through the glass tubing projecting from the proximal end of the bronchoscope down to the narrowed proximal end of the glass tube of the balloon, where it cannot go farther. The wire is firmly held at this point and the glass tubing is pulled gently in order to dislodge the balloon from the rest of the system. The balloon is now at the desired site and firmly held. The bronchoscope is removed, and the animal is ready for further observation.

This method of bronchial obstruction is the first of its kind, so far as we know. Traube and Mendelssohn used paper plugs and Lichtheim sticks of laminaria. They introduced these obstructions through an opening in the trachea by long, curved forceps.

The filling up of the balloon with an opaque solution allowed us to locate it easily under the fluoroscope and to measure its diameter. Serial roentgenograms were taken of all our animals.

PHRENICOTOMY

We have not been able to produce atelectasis of the lungs by section of one or both phrenic nerves. The corresponding diaphragm under the fluoroscope was higher than the other and immobile, sometimes presenting slight movements. We often noticed the "paradoxical movements" described by Lemon, i. e., the ascension of the diaphragm with inspiration and its descent with expiration.

In one dog (210) the pneumographs were connected so as to register on the drum any changes in the respiration during operation. The pneumographs were fixed on the animals with hooks screwed into one spinal apophysis and into the sternum, as previously described. We give the protocols of three experiments, dogs 211, 216 and 217. In the first, 211, both phrenic nerves were sectioned, the right first, and the left five days later. The respiratory tracings were taken during the operation. The times of pinching and cutting the nerve were recorded. Special disturbance was not noticed in the respiratory rhythm of the affected side. The first operation was conducted under ether anesthesia (the iso-amytl-ethyl barbituric acid given was insufficient) administered by slight intratracheal insufflation. The second operation was carried on solely under iso-amyl-ethyl barbituric acid anesthesia.

No changes were noticed during the pulling, crushing or cutting of the nerve and no differences between the respiratory rhythms of the two sides.

EXPERIMENT 1 (dog 211).—A white male terrier, weighing 6 Kg., was used.

Iso-amyl-ethyl barbituric acid, 10 per cent solution, was given intraperitoneally, 55 mg. per kilogram of body weight, or 3.3 cc. After twenty minutes, ether was given by the intratracheal tube as the anesthesia was insufficient.

The pneumographs were fixed on the back and the sternum of the animal with hooks screwed into the spinal process of the seventh thoracic vertebra and the

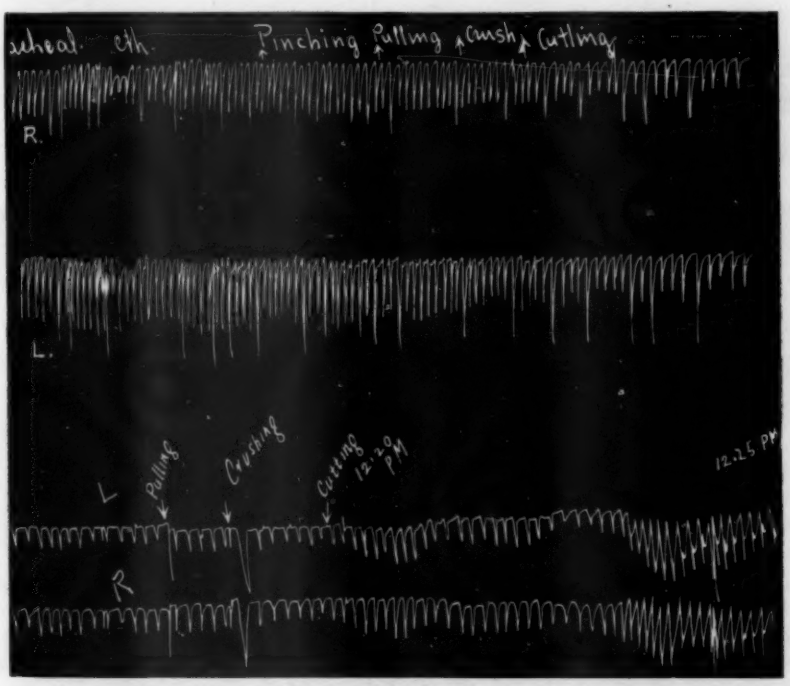

Fig. 1 (dog 211).—Double pneumograph tracing. The upper figure shows resection of the phrenic nerve under ether anesthesia; no changes were noticed during the pinching, pulling, crushing or cutting of the nerve, and no differences in the respiratory rhythm between the two sides. The lower figure shows resection of the phrenic nerve (*L*) of the same dog four days later; no changes were noticed. (Figures 1, 2 and 3 have been retouched.)

junction of the middle and inferior third of the sternum. The pneumographs were connected with the tambours so as to take tracings during the operation.

An incision was made in the skin behind the posterior border of the right sternomastoid, which was held mesially. The phrenic nerve was found; its roots were cut, and every branch which was thought to anastomose with the trunk of this nerve was cut. The nerve was pulled out and avulsion performed.

Changes were not observed in the tracings (fig. 1) during the operation. The nerve was pulled or pinched without any noticeable difference in the tracing, and the avulsion did not produce any difference in the curves.

The muscular layer of the aponeurosis and the skin was sutured with fine silk.

By Feb. 20, 1927, the dog made an uneventful recovery, without any respiratory trouble. The only reactions on auscultation or percussion of the right side of the thorax were signs of a slight elevation of the diaphragm.

On February 23, the animal was lively, and ate and drank well; the temperature and pulse were normal, and there was no dyspnea.

On February 24, iso-amyl-ethyl barbituric acid, 66 mg. per kilogram of weight, or 3.8 cc. of 10 per cent solution was injected into the peritoneum. Complete and deep narcosis occurred after twenty minutes. Pneumographs were made and phrenicotomy was performed on the left side with the same technic as in the previous operation. Changes were not observed in the tracing (fig. 2).

On March 3, an abscess developed in the back, at the place where the hooks were fixed. It was opened and dressed.

On March 16, the animal had recovered. Nothing abnormal was noted in the lungs. The dog was killed with ether.

Autopsy.—The lungs were in perfect condition, except for a slight congestion on both sides. Collapse did not occur. Both lungs as well as small pieces cut off

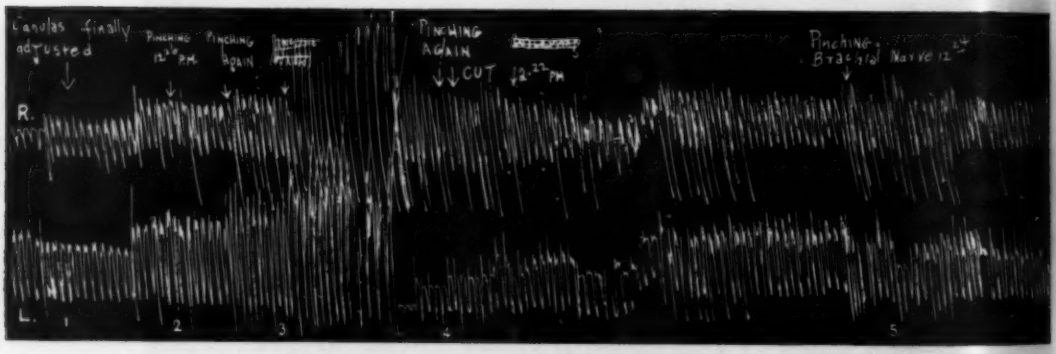

Fig. 2 (dog 216).—Tracing showing respiratory movements when phrenicotomy was performed on the right side; intrapleural pressure during operation. *1.* Intrapleural cannulas were adjusted. *2.* and *3.* Pinching produces a slight increase in respiration, apparently more marked on the left side, but immediately afterward, the movements become equal on both sides. *4.* Cutting of phrenic nerve is not marked by any disturbances in respiratory rhythms. *5.* Pinching branchial plexus produces an increase in respiratory movements; there is no difference between the intrapleural pressure of each side.

at the congested areas of the bases floated on the surface of water. Both phrenic nerves were cut down to the thoracic cavity (retraction after avulsion). Both diaphragmatic domes were pronounced and of equal height. The diaphragm was extremely thin, like a thin grade of paper, especially in the center; but even in the periphery it looked much thinner than normal, especially on the right side.

The intrapleural pressures recorded during section of the phrenic nerve of one side do not disclose any difference between the sides. In two experiments (216 and 217), we noticed violent respiratory movements and variations in the intrapleural pressures on pinching the phrenic nerve, owing to the superficial anesthesia. Both animals had

been given iso-amytl-ethyl barbituric acid. These disturbances disappeared in a few minutes. We did not notice any difference between the two sides.

EXPERIMENT 2 (dog 216).—A white cocker-spaniel, weighing 7.7 Kg., was used. On March 7, 1927, iso-amyl-ethyl barbituric acid, 58 mg. per kilogram of weight, or 4.4 cc. was administered; fifteen minutes later, anesthesia was complete, but not extremely deep. At the fifth interspace on the right side at the axillary line, a small incision (1 mm.) was made in the skin, and the intrapleural cannula, connected by a sterile rubber tubing with a tambour, was forced into the intrapleural space. All aseptic measures were taken. The lever of the tambour immediately started to oscillate, which was proof that the right space had been entered and that pneumothorax was not present. The same procedure was repeated on the left side. Each intrapleural cavity was connected with a tambour. Phrenicotomy was performed on the right side with the usual technic. When the nerve was pulled or pinched, a violent respiratory movement occurred. After a few seconds, the respiration became almost normal. This disturbance in the rhythm seemed to be due to the reflex produced by the pinching, because it was

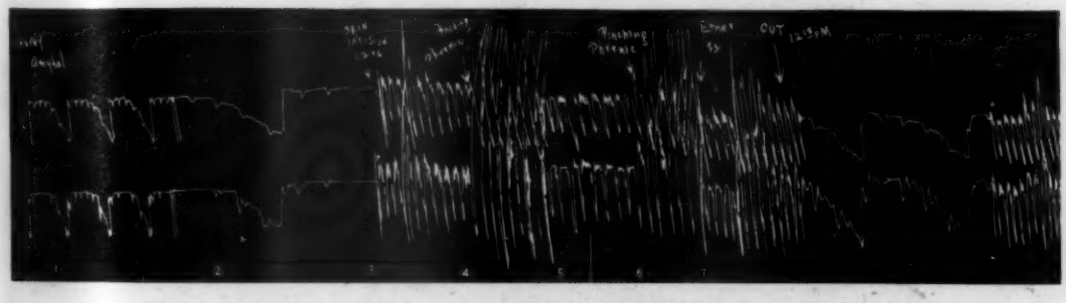

Fig. 3 (dog 217).—Tracing showing respiratory movements during intrapleural pressure. *1.* Regular respiration; excursion on both sides equal; upper is right side and lower is left. *2.* Slight decrease in respiratory excursion has often been noticed without any apparent cause, and is of temporary duration, 12:30 p. m. *3.* Incision of skin; dog reacts slightly; respiration increased. *4.* Pinching of right phrenic nerve; straight respiratory movements on both sides. *5.* Respiration back to normal. *6.* Pinching again; same reaction. *7.* Right phrenic nerve cut, 12:18 p. m.; two long respiratory movements followed by rapid respiration again.

reproduced when we crushed the brachial plexus of the animal. "Collapse" did not follow (fig. 2).

The same phenomenon was produced in dog 217 under the same circumstances. Phrenicotomy was performed on the left side. Collapse did not follow (fig. 3).

In order to find out what would happen if an occlusion of the bronchus was performed when the diaphragm was previously paralyzed by section of the phrenic nerve, we performed a phrenicotomy on the left side on dog 242 under iso-amyl-ethyl barbituric acid anesthesia. A roentgenogram taken before phrenicotomy showed clearly that the

right side of the diaphragm was higher than the left (fig. 4); whereas in the following roentgenogram (fig. 5), which was taken one-half hour after phrenicotomy was performed on the left side, the left side of the diaphragm was shown to be higher than the right; under the fluoroscope, the left side showed little mobility and paradoxic movements. Five days later, the right lower bronchus was blocked with our balloon, which was filled up with saturated solution of potassium bromide so as to make it visible. In a roentgenogram (fig. 6) taken twenty-two hours after the blocking, "collapse" of the lower right lobe was apparent; the heart had shifted toward the right side and the right

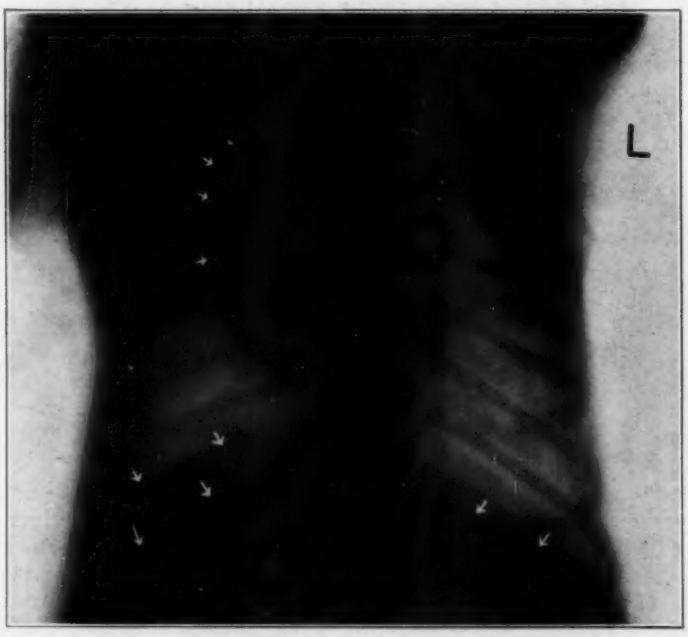

Fig. 4 (dog 242).—Roentgenogram taken before phrenicotomy was performed on the left side; double exposure on the same film; inspiration and expiration; right side of the diaphragm is higher than the left; excursion of the diaphragm is indicated on the right side; left diaphragm is immobile.

side of the diaphragm was much higher than the left. This showed clearly that the ascension of the diaphragm followed the "collapse," even when the other side of the diaphragm was paralyzed. The dog died two days later. Autopsy revealed that section of the left phrenic nerve had been complete and that the right side of the diaphragm was slightly higher than the left. The lower and middle lobes of the right lung had collapsed and were a dark blue-black and of leathery consistency; these lobes sank in water, and if compressed while under water, did not give off air bubbles. The balloon was in place, and partially obstructed the bronchus of the upper lobe.

The same phenomena were produced in all our experiments. The diaphragm is always higher on the side on which atelectasis has occurred than on the sound side.

2. The most important part of our experimental work has been directed to the study of the obstruction of the bronchus and its results. First of all, we tried to find a way by which it would be possible to follow the successive stages of atelectasis by direct vision. We tried the transparent thoracic window without great success, using cats for the experiments. The thoracic cavity was opened wide, and a plate of

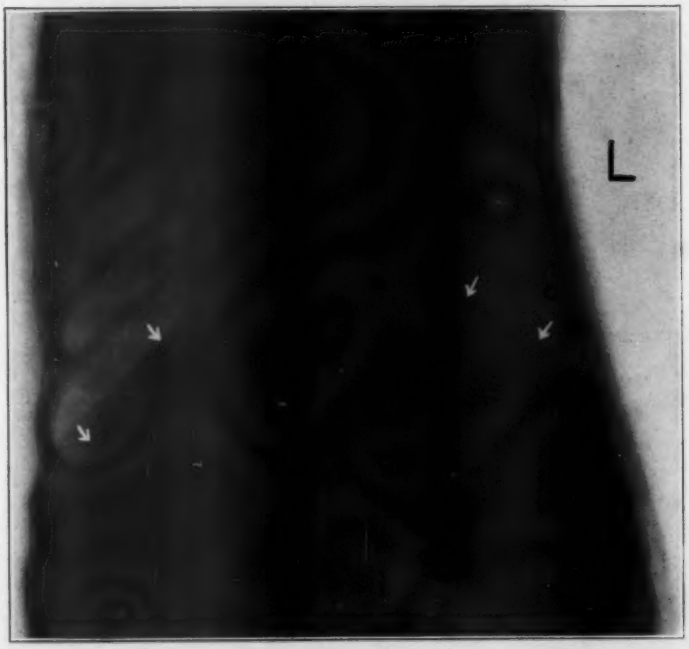

Fig. 5.—Roentgenogram taken one-half hour after phrenicotomy has been performed on the left side. The left side of the diaphragm is higher than the right.

celluloid was shaped so as to occlude the opening of the thorax and to avoid desiccation of the lung. A bronchus was then occluded by the bronchoscopic introduction of our balloon. The impossibility of maintaining the celluloid in good position for several hours, as was necessitated by our experiment, obliged us to search in another direction.

We thus adopted our "vacuum box" (fig. 8), which was constructed under our direction. This is composed of an air-tight wooden box measuring 32¾ inches (83.17 cm.) in length, 14 inches (35.48 cm.) in width and 12 inches (30.48 cm.) in height; it is covered by a glass top which runs in a groove in the frame and thus permits the box to be

closed hermetically. The groove is filled with mercury or preferably with petrolatum, so that a perfect seal is obtained. The box has three outlets; two of them are 1.3 cm. in diameter and the third, 0.3 cm. The first outlet is connected with a rotating valve (*V*) which produces an oscillating vacuum. The valve is rotated slowly by a home-made two-pulley speed reducing mechanism consisting of a wheel (*W*)

Fig. 6 (dog 242).—Roentgenogram taken twenty-two hours after obstruction of the right lower lobe with balloon; balloon is filled with bromide solution; the heart shifted toward the right; the right side of the diaphragm is higher then the left which is paralyzed; atelectasis of the right lung.

and a motor (*M*) with a rheostat installed. An extra 40 watt lamp resistance (*L*) in the system further reduces the speed of rotation. The valve can be regulated to perform only from fifteen to twenty revolutions a minute, simulating the respiratory movements. The

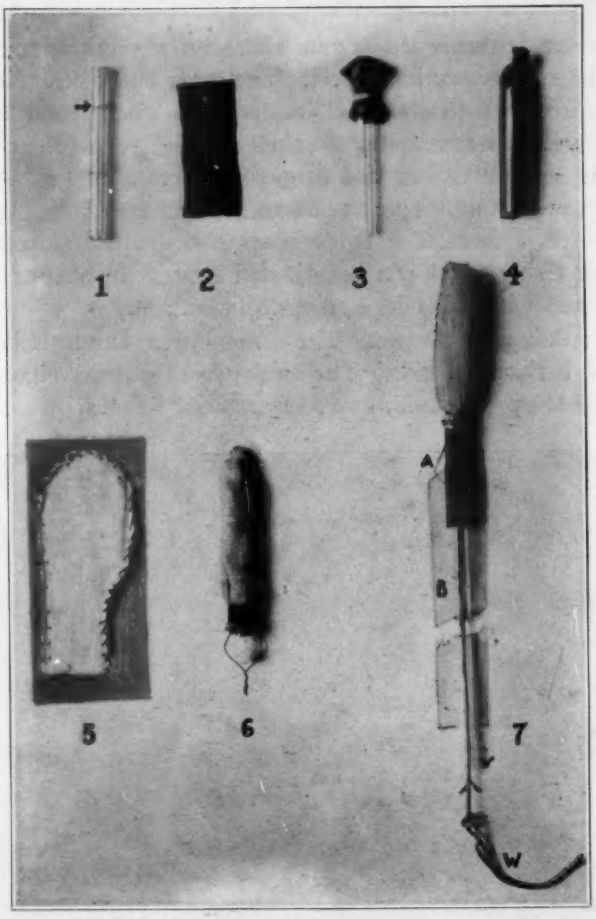

Fig. 7.—The occluding balloon is constructed with: *1*, a glass tube 4 cm. long and a little less than 0.5 cm. across (there is a slight narrowing where the arrow points; the lower opening is almost punctiform); *2*, a piece of old rubber glove a little more than 1 cm. by 3 cm.; *3*, the rubber tissue tied over the upper end of the glass tube under slight tension, forming a one-way valve; *4*, a piece of Kollman rubber tubing 4 cm. long into which the glass and valve are introduced; *5*, a more or less elliptical covering of fine organdie which is slipped over the glass, valve and tubing of the previous stages (it has a potential diameter of 1.5 cm. or more). In *6*, the organdie covering has been twisted, expelling the air within the system and diminishing its volume. The rubber tube and its covering are now tightly tied 1.5 cm. from the lower end of the tube. Superfluous rubber and material are cut away. A piece of fine piano wire is wound around the neck of the balloon in the shape shown, to hold the balloon in place and facilitate its removal. In *7* is shown a schematic representation of a blown balloon which is ready to be removed and left in a bronchus. The balloon is attached at point *"A"* to the long glass tubing shown projecting through the bronchoscope *"B."* Wire *"W"* is put through this glass tube as far as it will go (point *A*). By holding the wire steadily at this point, and pulling on the glass tube, the balloon is detached. Bronchoscope, tubing and wire are now removed from the trachea.

rotating valve is connected at one end with a suction machine or the ordinary laboratory suction faucet. The other end of the rotating valve connects directly with the box (outlet *1*). The second tube (*2*) is connected with a water manometer. The third tube (*3*), covered with a piece of rubber tubing and stop-cock, connects the box with the outside air, regulating the degree of vacuum in it. At the opposite panel of the box is a circular opening bearing a cylindrical collar (*C*) of soft rubber tissue which can be adjusted around the shaved neck of the dog and fixed with a bandage so as to be airtight.

Our technic was simple. The animal was anesthetized with iso-amyl-ethyl barbituric acid. The neck was then shaved, the anterior wall of the chest prepared, and intratracheal insufflation started. The

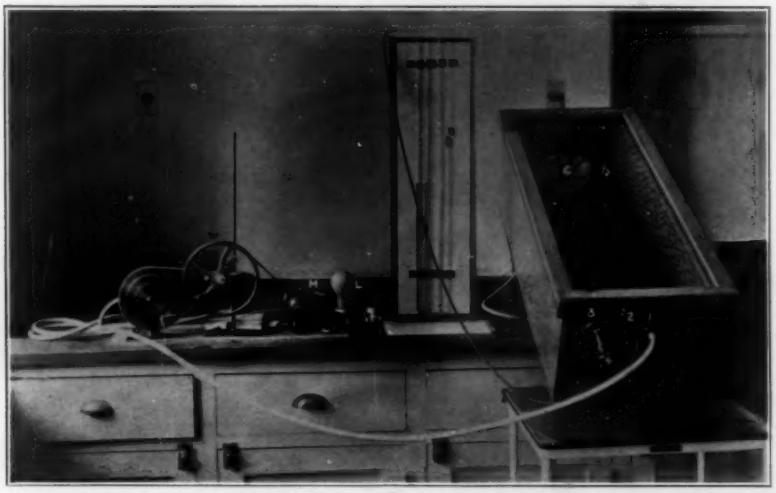

Fig. 8.—Box for oscillating vacuum. *1, 2* and *3* indicated outlets; *V*, rotating valve; *W*, wheel; *M*, motor; *L*, 40 watt lamp resistance and *C*, a cylindrical collar.

thorax was then opened by hemisection of the sternum as in the technic of Gregoire, and a Balfour retractor was applied to maintain the thoracic cavity wide open. As there is but one pleural cavity in the dog, physiologically speaking, it did not matter if both cavities were exposed. The animal was then introduced into the box through the circular opening or through the top of the box, as temporary interruption of respiration does not have any evil effect. The cover was tightly applied and the rubber collar fixed around the neck of the animal, smeared with petrolatum and secured by a gauze bandage. Care was taken not to exert any unnecessary constriction around the neck.

The suction faucet was opened and the rotating valve set in motion. By regulating the opening of the suction faucet and of outlet 3 of the box, we easily obtained a negative pressure oscillation between 3 and

6 cm. of water, with from eighteen to twenty revolutions a minute. This proved sufficient to keep the animal alive for several hours (up to six hours in dog 214 when the suction was cut off by accident while the heart was beating regularly and strongly, and twelve and a half hours in dog 224). When the animal was thus placed and the intratracheal tube withdrawn, it breathed normally by mouth, exactly as if the intrapleural pressure had not been disturbed. Curiously enough, with a rate of eighteen revolutions a minute of the valve, dog 214 had real respiratory movements of its thoracic walls at one-half the rate of the revolutions of the valve. When the rate of the valve was increased, the respiratory movements slowed down. It was thus possible to reach at least a temporary equalization of the two rates. After regulation of the respiration, the bronchoscope was introduced and a bronchus blocked.

EXPERIMENT 3 (dog 218).—A white male poodle, weighing 7.7 Kg., was used. Iso-amyl-ethyl barbituric acid, 60 mg. per kilogram of weight, was injected into the peritoneum. Anesthesia was complete in fifteen minutes.

March 10, 1927, 10:25 a. m.: Intratracheal insufflation was established. The thoracic cavity was opened by midsternal section (Balfour retractor). The animal was placed in the negative pressure box and the collar fastened around its neck. The cover was replaced and the box tested air-tight. The box was connected with the rotating valve and regulated to eighteen revolutions a minute and negative pressure oscillations between 3 and 7 cm. of water. The insufflation was disconnected. The lungs dilated regularly in synchronism with the revolution of the vacuum oscillations, but the animal made independent respiratory movements from time to time.

12:05 p. m.: A balloon was introduced through the bronchoscope into the inferior right lobe bronchus and blown up with 5 cc. of water so as to occlude the lobe.

12:15: The lobe shrank to half its size and was yellow-pink, slightly darker than the rest of the lung; it did not expand during inspiration.

12:30: The condition was the same. The right lung was darker and the lower lobe was bluish. The heart beats were strong and regular.

12:45: The lobe became darker and the size of the lung continued to diminish.

1:30: The inferior lobe was markedly blue, and dark bluish-black patches appeared on its surface. The vacuum was purposely shut off for a few seconds. The other lobes became completely disinflated; the occluded lobe did not. This showed that the obstruction was good.

2:05: The heart gradually stopped beating. After the injection of epinephrine into the heart, it was revived for a few moments and then stopped definitely.

Autopsy.—The lower right lobe was bluish with dark patches on its surface and was one third of the normal size, the trachea being clamped. It was not completely without air, but a piece of the lobe barely floated in the water; the other lobes floated readily on the surface.

EXPERIMENT 4 (dog 219).—This experiment was the same as that performed on dog 218, with the difference that the thoracic opening was occluded as completely as possible with a sheet of celluloid uniformly shaped and introduced under the borders of the wound. Wet pads were placed in the box to avoid desiccation.

11:50 a. m.: The animal was placed in the box. As soon as the vacuum was established, the rhythmic expansion of the lungs started, filling the thoracic cavity completely with each inspiration. The rate of the valve was 18 a minute; the spontaneous respiratory removements of the animal, 9 a minute. The valve rate was increased to 24 a minute; the spontaneous respiratory movements slowed down to 5. By changing the valve rate to 12, we obtained a synchronization with the spontaneous movements of the chest.

12:30 p. m.: The right main bronchus was blocked with a balloon.

1:00: The size and color of the right lung did not change. A second balloon was introduced and inflated. Immediately there was marked shrinkage in the upper and middle lobes. The left lung (distended) took the place of the shrunken lobes and pushed the heart to the right side. Apparently the first balloon was not sufficiently inflated. When the second balloon was introduced, it seemed to impinge against the orifices of the two superior secondary bronchi, whereas the first balloon, which was placed near the internal wall of the main bronchus, prevented the complete contact of the second balloon with this internal wall; thus complete occlusion of the bronchus of the lower lobe was impossible.

1:30: There was marked deflation of the two upper lobes. Dark blue spots formed on the surface of the lung. Marked dyspnea was not present; the spontaneous respiratory movements of the animal were not increased in number.

2:00: The upper lobes were still more shrunken and the dark blue spots became more and more confluent.

5:00: The heart beat was strong. The manometer oscillated between 1 and 4 cm. of water. The upper and middle lobes of the right lung were much shrunken and immobile. The heart was displaced more to the right.

6:00: The suction apparatus suddenly went out of commission. The animal died.

Autopsy.—The upper and middle right lobes were shrunken and bluish-black, the trachea being clamped. The left side of the lung was normal. The lower right lobe was slightly cyanotic, but normal in consistency. On palpation, the resistance of the two upper right lobes increased and very few air bubbles came out on compression as compared to the other lobes; pieces of the upper and middle right lobes barely floated, whereas pieces of the other lobes floated readily. The lower balloon was found disinflated, substantiating our surmise.

EXPERIMENT 5 (dog 224).—A mongrel, male terrier, weighing 9 Kg., was used.

March 21, 1927, 10:40 a. m.: The animal was put to sleep with iso-amyl-ethyl barbituric acid, 70 mg. per kilogram of body weight.

12:45 p. m.: Midsection of the sternum was performed, which was then held closed with clamps.

1:45: The animal was placed in the box.

1:55: By means of an aspiration tube placed through the bronchoscope, oxygen was introduced under pressure into the right bronchus for a period of over five minutes.

2:00: A balloon was introduced into the right lower bronchus. The animal breathed synchronously with vacuum even when the rate was changed. The clamps on the chest were removed every hour, and the condition of the lungs was noted.

3:00: The right lower lobe seemed to be very much shrunken. The left lobes were much larger and extended further toward the right side than previously.

4:00: The heart beat was strong. The rate of respiration was 2:1 of the vacuum rate. The right middle lobe was not seen. The right lower lobe seemed to be shrunken and almost hidden behind the heart.

5:00: The heart beat was strong, the rate moderate; the respiration rate was 32 and the vacuum rate, 16. The right lower lobe was bluish and well hidden behind the heart, close to the vertebral column.

6:00: When the clamps were removed and the chest opened, the animal made vigorous movements of the chest, which made it extremely difficult to keep the thorax open. The chest was quickly clamped and the cover of the box tightly sealed. The heart beat was strong, and the dog breathed well.

7:00: The respiration rate was 32; the vacuum rate, 16. The right lower lobe was small and was seen with the greatest difficulty by retracting the heart to the left.

8:00: A slight amount of ether was administered by the bronchoscope. The left lobes were prominent, especially the lower lobe. The right lower lobe was the same as before. The vacuum rate was 18; respiration in the vacuum box was 22; respiration without the vacuum was 28, the chest being clamped.

9:00: The animal struggled violently when the chest was opened. Further marked changes were not seen. The anesthesia was taken well.

10:00: The manometer read 0 to 2.

12:00: The dog was in good condition and breathed well. There were no more changes than at the previous examination.

March 22, 1:20 a. m.: A sufficient amount of ether was administered under slight pressure. Changes were not noted.

1:45: The vacuum apparatus went out of order, but the animal breathed well with the chest clamped. The manometer read 0 to 2.

2:30: The animal died.

Autopsy.—The trachea was clamped. The clamps were removed from the chest. The appearance was typical of massive atelectasis. The heart seemed to be to the right; the right side of the diaphragm was rather cupped and high. The left lobes were prominent and clearly seen, although one had to push the heart to the left to see the right lower and accessory lobes, which were shrunken and of a livid hue. The right lower lobe was affected in its lower two-thirds. The affected lobes sank in water, were airless and firm.

Microscopic section of the affected lobes revealed complete atelectasis without complications.

These three experiments show clearly the gradual production of atelectasis by obstruction, the development of which we followed step by step. Atelectasis was not complete in the first two cases because the time was not sufficient, but it was complete in the third case, in which the lobe was filled with oxygen. It was difficult to keep the animal alive under these conditions; several animals died within from one to three hours after the beginning of the experiment. The same sequence of phenomena always presented itself. Although the observations made during these experiments were convincing, we believed that it was necessary to find out what happens when the lung is occluded under normal conditions when the pleural cavity is intact.

3. Obstruction of a main or secondary bronchus was carried out in thirty-three dogs; fluoroscopies were carried out and serial roentgenograms made; clinical evolution was closely followed, autopsies recorded and sections of the lung examined. In all our cases, we reproduced

the typical symptoms of atelectasis (apneumatosis), i. e., dyspnea, cough, diminution of normal resonance on the affected side progressing to dulness and shifting of the mediastinum toward the same side, with a more or less marked rise of temperature. The production of the condition is so typical after the complete occlusion of a lung that when it was not produced, we were certain that something was wrong with the balloon and that obstruction was not complete. The case of dog 232 proves this.

EXPERIMENT 6 (dog 232).—A female fox terrier, weighing 10 Kg., was used.

May 26, 1927, 10:30 a. m.: Iso-amyl-ethyl barbituric acid, 65 mg. per kilogram of weight, was administered.

10:45: The dog slept deeply. A roentgenogram of the chest was taken.

11:00: The right main bronchus was blocked and 7 cm. of saturated potassium bromide solution was injected into the balloon.

11:15: A roentgenogram was made. Clinically, the heart did not shift nor was there any displacement of the heart toward the right side. In order to find out whether the balloon was inflated or not, a lateral roentgenogram was taken, which showed that the balloon was not inflated.

12:10 p. m.: The balloon was extracted through the bronchoscope. A new balloon was introduced into the right bronchus. A lateral roentgenogram was taken, which showed that the balloon was well blown up.

1:00: A clinical examination showed that the heart had shifted to the right, and that there was dulness at the base on the right side, with absence of vesicular murmur. There was tubular breathing over the upper part of the thorax on the right side, accompanied by wheezing.

5:00: Five hours after blocking, opacity of the lower right lung, displacement of the heart to the right and ascension of the right side of the diaphragm were noted.

4:00 a. m.: On May 27, sixteen hours after blocking, atelectasis increased. The heart was not visible to the left of the sternum. The animal was dyspneic, with complete dextrocardia.

3:30 p. m.: The balloon was removed. A roentgenogram taken ten minutes after removal showed the right side partly cleared up and the heart again markedly visible on the left side. The right side of the diaphragm was still higher than the left.

10:00: Six and a half hours after removal, the right side of the thorax was clear, the heart had come back to its normal position and the right side of the diaphragm was almost at the same level as the left. The clinical symptoms had completely disappeared.

May 28, 10:30 a. m.: Nineteen hours after removal of the balloon, recovery was complete. The diaphragm was at the same level.

In dog 246 (figs. 9, 10, 11), atelectasis was clearly shown by the three roentgenograms. Complete occlusion of the left bronchus was demonstrated by iodized oil 40 per cent injected into the trachea; none of the oil penetrated the left bronchus. The base of the right side of the lung had encroached on the left side, and traces of the oil were visible.

Fig. 9 (dog 246).—Roentgenogram taken before blocking.

We do not think it possible to have a more convincing experimental proof of the relation between obstruction of a bronchus and atelectasis of the corresponding side of the lung, and of the necessity of having this occlusion complete. The experiments also show that atelectasis disappears as soon as the obstructing agent is removed. The same

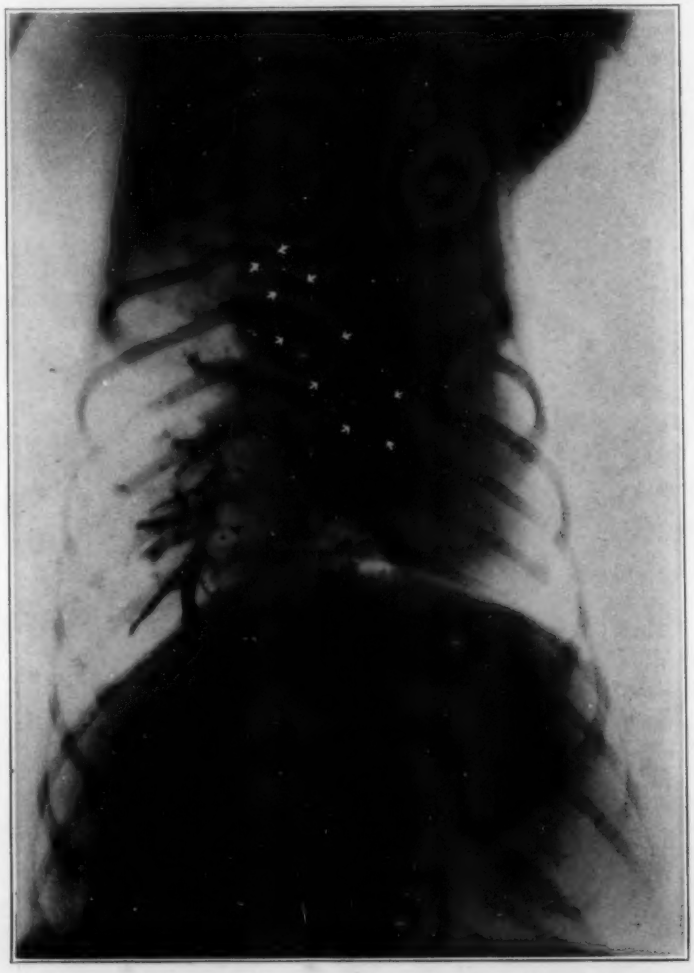

Fig. 10 (dog 246).—Roentgenogram taken twenty-five minutes after blocking; 2 cc. of iodized oil 40 per cent was injected into the trachea above the balloon by bronchoscope. Note that none of the iodized oil has entered the left bronchus, the right lower lobe has encroached on the left base, the diaphragm is high on the left and the shifting of the heart.

phenomena are reproduced when the balloon is disinflated slowly and spontaneously because of a small leak at its neck. Roentgenograms of dog 255 (figs. 12, 13, 14) show this clearly.

The possibility of establishing atelectasis of the lung with an intact thorax having been demonstrated, we continued our investigation further. The problems to which we tried to find the solution were:

1. The rapidity of the establishment of atelectasis when the lung is filled with oxygen before the obstruction.
2. The complications of atelectasis.

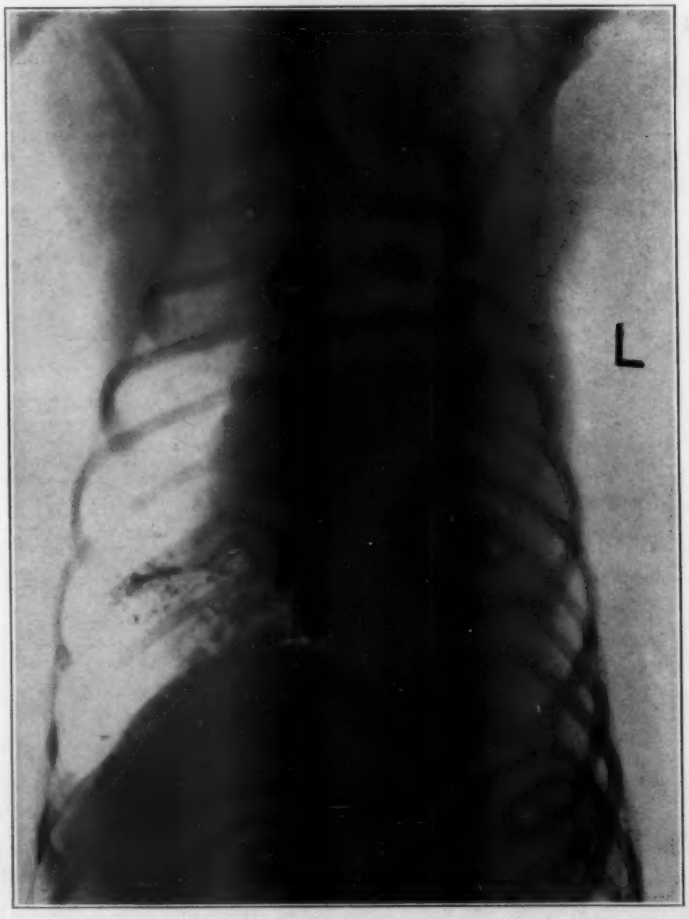

Fig. 11 (dog 246).—The animal died fifteen hours after blocking. Roentgenogram was taken six hours after death. The heart is greatly dilated; no iodized oil in the left lung; right diaphragm higher than during life (fig. 10).

3. The result of the extraction of the obstructing balloon in the complicated cases.
4. The relation between atelectasis (apneumatosis) and localization of the infection in cases of bacteremia.
5. Pathologic processes in atelectasis obtained by obstruction and the gross and microscopic complications.

Space does not allow us to deal with these questions in detail; they will be elaborated in subsequent papers. We shall give briefly the results of our investigations, which we are continuing, especially those made to localize the infection in the atelectatic lung.

1. The time necessary for the establishment of atelectasis when the lung is filled up with oxygen is short. Immediately after the occlusion, the heart is markedly shifted, and after one-half hour the atelectasis seems complete. Atelectasis is a much slower process when there is air in the lungs, although by roentgen-ray examination the process is marked by the sixth hour.

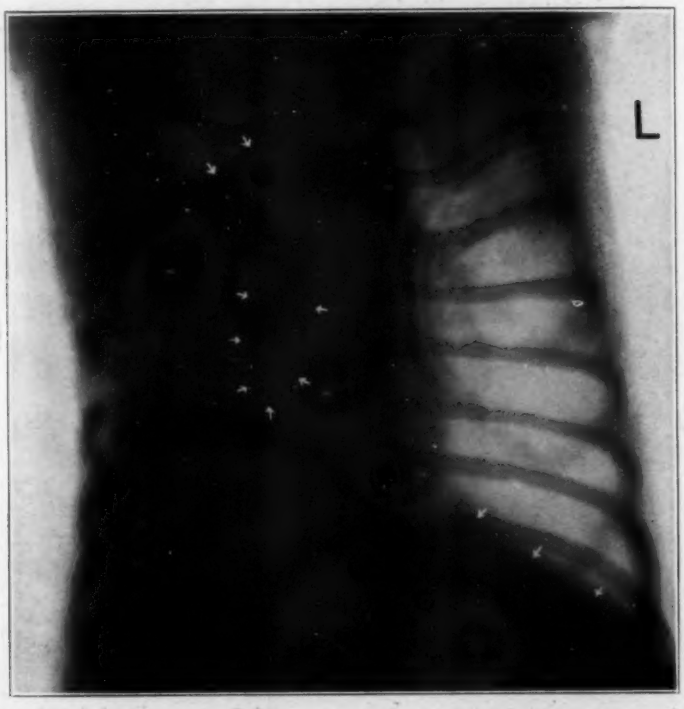

Fig. 12 (dog 255).—Roentgenogram taken twenty-four hours after blocking; atelectasis of the right lower and median lobes is noted; balloon is well blown.

2. The most frequent complication of the condition is a diffuse suppuration—a real purulent pneumonitis of the lung, bronchial in origin. Other types recognized in our sections, which were examined by Drs. L'Esperance and Symmers, were: acute exudative pneumonia, acute hemorrhagic pneumonitis (as in influenza) and typical bronchopneumonia. The most frequent agents in this infection were staphylococcus, streptococcus and especially *B. coli* and anaerobes.

3. Even when infection of the bronchus seems advanced and the animal is already in poor condition with a temperature as high as

105 F., recovery may occur, if the balloon is extracted and the pus contained in the affected bronchus aspirated. Recovery is impossible only in extremely severe cases, when the animal is moribund.

The time of the development of infectious complications is variable. It varied from twenty-four hours to six days in our dogs. Complications seem inevitable when the occlusion remains complete for more than seventy-two hours. We often recorded death occurring before the fourth hour, probably due to heart failure or hypertoxic infection.

Fig. 13 (dog 255).—Roentgenogram taken five days after blocking; only the right median lobe is slightly hazy; balloon is less inflated.

Death occurred immediately only in rare cases in which the balloon, displaced from a main bronchus, occluded the trachea and killed the animal by asphyxia.

4. The relation between atelectasis and the fixation in the lung of circulating microbes is one of the most interesting features of this postoperative complication. Cutler and his associates, Scott, Elwyn and others, especially Karsen (Scott), believed that apneumatosis favors the development of bronchopneumonia, and, more rarely, pneumonia and

even abscess of the lung, because of the fixation of septic emboli in the tissue of the affected lung, which would not produce complications in a sound lung. Our investigation of this problem is not yet finished. It presents important technical difficulties, chiefly because of the suppurative pneumonitis which, as a rule, follows occlusion in the dog, probably because of *B. coli* and other organisms (present in the mouth and pharynx) which the balloon carries down into the lung. We will give here only two roentgenograms (figs. 15, 16) of dog 251 in which

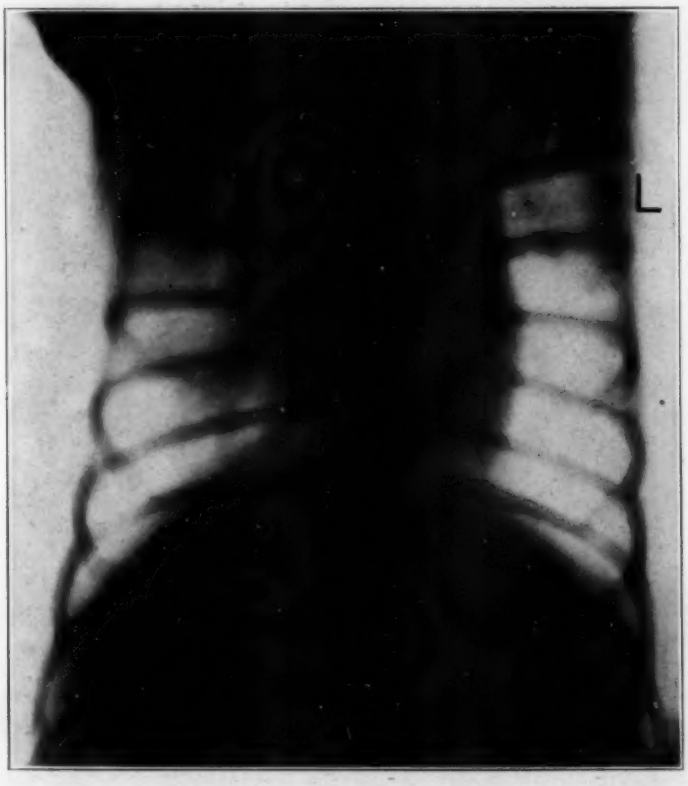

Fig. 14.—Roentgenogram taken twenty-four hours after the balloon was coughed up; the right lung is almost entirely clear.

the lung showed a diffuse suppurative bronchopneumonia after intravenous injection of a special *B. coli* strain after blocking. The *B. coli* cultured from pus in the small bronchioles was not identical with the culture injected and corresponded to the *B. coli* normally present in the mouth and pharynx of the dog. The cultures were supplied and the bacteriologic work controlled by Drs. J. C. Torrey and M. C. Kahn.

5. The gross and microscopic pathologic processes in our experimental atelectasis are typical. Figure 17 is a gross photograph taken

immediately after autopsy on dog 254. The nonatelectatic lung seems small because the clamp occluding the trachea was taken off by mistake before the photograph was taken. With the trachea clamped, the healthy lung was two times the size of the affected lung, which had a blackish color and a liver-like hue. The bronchi below the balloon were filled with mucus and often with pus. We have never found the bronchi below the obstruction free from secretion.

Fig. 15 (dog 251).—Roentgenogram taken forty-eight hours after injection of second doses of anaerobes; lungs clear; no abscess.

Adhesions of the pleura were found in one case, and foul effusion in the pleura was present in another. Patchy "collapse" was present in three cases in which the bronchioles were filled with pus. Bilateral pleurisy was noticed in two cases. We had no case of lobar pneumonia, although we had several in which edematous or congested lungs with areas of alveoli filled with serum and leukocytes were suggestive of pneumonic consolidation (fig. 18).

The microscopic aspect varies with the degree of infection superimposed on the "collapse," but in our experiments it invariably showed complete disappearance of the alveolar spaces (fig. 19) comparable to the fetal lung (fig. 20); marked dilatation of the capillaries, which were filled up with blood; bronchioli filled with exudate, serofibrinous or purulent, and edema of the capillary wall. Often there was infiltration

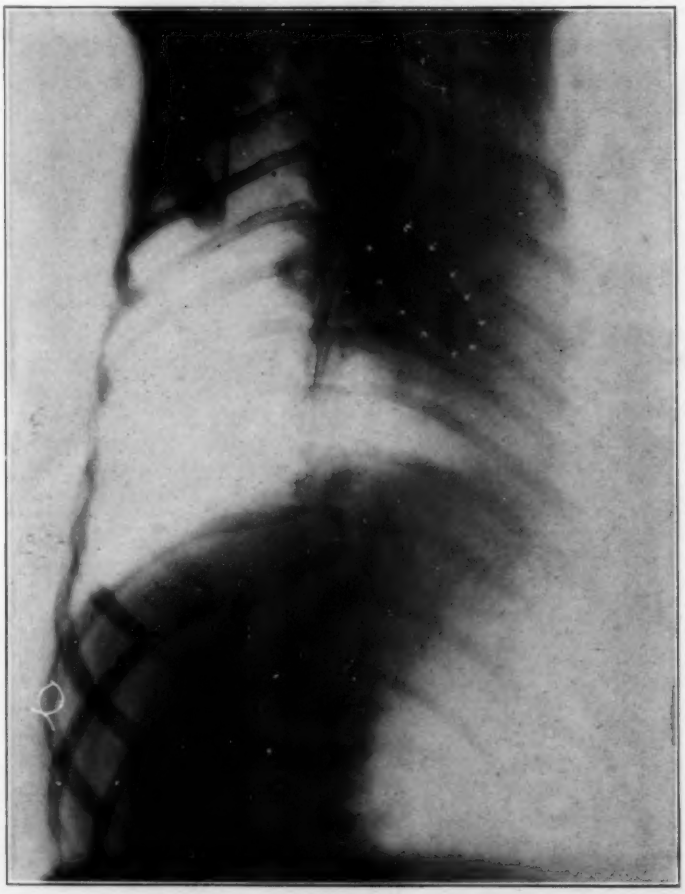

Fig. 16 (dog 251).—Roentgenogram taken six days later; left lung was blocked and 1 cc. of *B. coli* culture injected intravenously; four days later the dog died and a nonspecific *B. coli-streptococcus* bronchopneumonia was found superimposed on atelectasis.

of the alveolar walls and the peribronchial spaces with polymorphonuclears which were confluent and gave the picture of diffuse suppuration. In a few cases, necrosis of the parenchyma was observed comparable to gangrene of the lung.

It is clear from the foregoing experimental results that there is not the slightest etiologic, clinical or pathologic difference between our

experimental cases and the human cases reported in the literature. We have purposely tabulated the latter to facilitate their comparison. We found 118 cases described and 12 mentioned in the literature, making a total of 130 cases. These do not include cases of frank obstruction by a foreign body. The observations made in our conclusions are based on this similarity, and, although they may appear

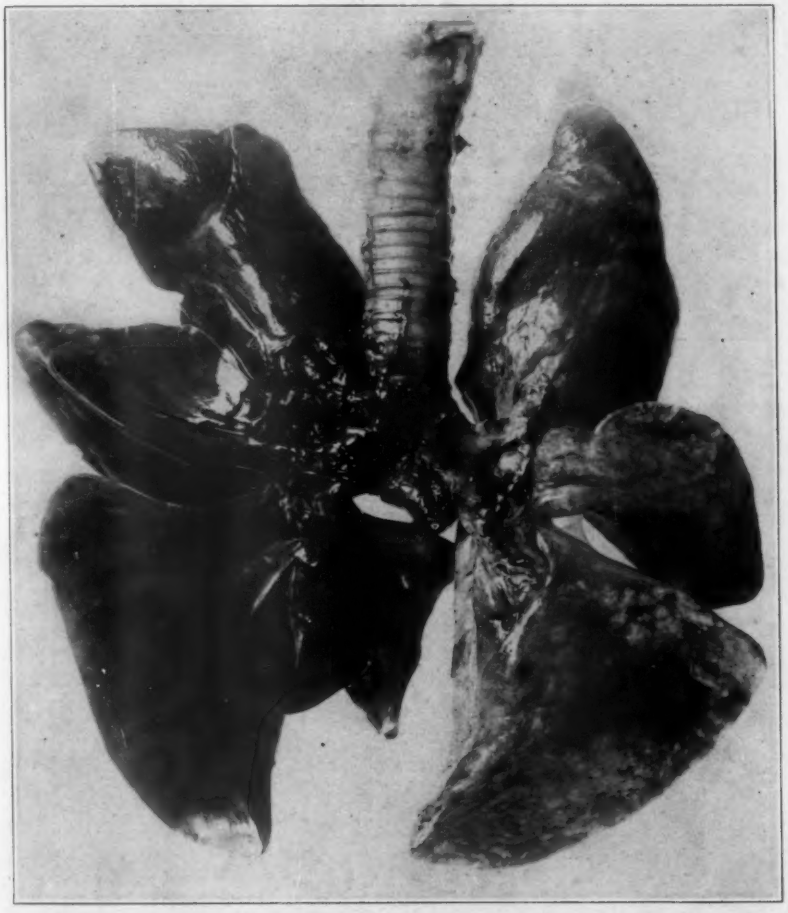

Fig. 17 (dog 254).—Massive collapse of the right lung; the clamp occluding the trachea was accidently removed before the photograph was taken. With the trachea clamped the healthy lung was twice the size of the affected one.

somewhat radical, we believe that they are backed by experimental and clinical proof. Above all, we think that a practical conclusion results from this study, namely, the importance in atelectasis of the removal of the occluding agent by bronchoscopy and aspiration, and the value of the therapeutic benefits that are obtained.

Fig. 18 (dog 199).—Photomicrograph showing one of the complications (acute exudative pneumonia) engrafted on an atelectatic lung.

Fig. 19 (dog 198).—Photomicrograph of an atelectatic lung. Compare with the fetal lung in figure 20.

The importance of bronchoscopic treatment and aspiration of secretion, exudate or membranes obstructing the lumen of a bronchus is apparent. Although the prognosis in this disease is generally good, one cannot know at the onset what its evolution, duration or complications will be. Therefore we cannot too strongly insist on the necessity of bronchoscopic treatment when shaking of the patient, change of position and other measures are without beneficial effect.

We suspect that under certain circumstances even fluids that are not very viscid may be able to "plug" a bronchus effectively; during our experiments, this question often occurred to us. The altogether

Fig. 20.—Photomicrograph of fetal lung (atelectatic lung).

special physical conditions existing in the bronchial tree (because of its shape), the constant decrease in the velocity of the currents of air and of the sensibility of the bronchial mucosa to cough, producing stimuli as the alveoli are approached (as pointed out by Lincoln and Archibald) and other factors are subject to wide changes and modifications with possible alterations in the mucosa and in its ciliary epithelium. Congestion, alteration in the diameter of the bronchial lumen, action of capillarity and other factors may render possible an effective occlusion of a bronchus by fluids of only moderate viscosity. The bronchial tree cannot be likened to a system of glass or rubber tubes, nor the alveoli to a system of rubber balloons. These are interesting problems, which we are investigating.

Statistics *

Writer	Complication or Forerunner; Location of Collapse	Age	Sex†	Deaths and Conditions Found Postmortem	Operation; Anesthetic	Onset; Symptoms	Duration	Treatment
Pearson-Irvine: Tr. Clin. Soc. Lond., 1876, p. 188	Diphtheria; right and left upper lobe	6	♀	.....	.....	4-5 weeks after diphtheria; flatness, dulness over both infraclavicular regions; pharyngeal paralysis, cough	14 days	
Pasteur, W.: Am. J. M. Sc. **100**: 242, 1890	Case 1: diphtheria; right and left lower lobe	4	♂	Collapse of right and left base; bronchi not described	.....	19 ? days after paralysis of the pharynx; 6 weeks after diphtheria	?	
	Case 2: diphtheria	4	♂	Death; no autopsy done	.....	5 weeks after diphtheria; palate paralyzed; cough on swallowing	?	
	Case 3: diphtheria; right lower lobe	4	♂	.....	.....	6 weeks after diphtheria; 6 days after paralysis of the palate; diminished resonance; weak bronchial sounds at base	?	Placed on left side with artificial respiration 3 times a day
	Case 4: diphtheria; both bases ?	2	♂	.....	.....	5 weeks after diphtheria; cough, cyanosis; temperature, 102.8	3 days?	Artificial respiration every 6 hours, 15 minutes for 3 days
	Case 5: diphtheria; whole right lung, especially lower lobe	2	♂	Collapse of right lung and almost all of lower lobe; bronchi not described	.....	7 weeks after diphtheria; gradual dyspnea, cyanosis	5 days? death sudden	Artificial respiration
	Case 6: diphtheria; right middle and lower lobe	..	♂	.....	.....	17 days after paralysis of the trunk and limbs; dyspnea, cyanosis; diminished expansion of chest	2 days	
	Case 7: diphtheria.....	6	♀	.....	.....	2 weeks after diphtheria; paralysis of the pharynx and larynx; began to cough	10 days?	
Buzzard, E. F.: Brain **28**: 458, 1905	Followed myasthenia gravis	48	♂	Collapse of base of left lower lobe; bronchi not described	.....	Choking fits; dyspnea befor death		
Pasteur, W.: (cont'd) Lancet **2**: 1351, 1908 "Bradshaw lecture"	Case 10: mitral regurgitation; failure of right side of the heart	48	♂	Partial collapse of both lower lobes; no bronchial obstruction	.....	Dyspnea, cyanosis, no dulness	?	
	Case 11: posterior part of right upper and middle lobe; right lower lobe; left lower lobe almost airless; followed operation and general peritonitis	49	♀	General peritonitis; right pleura, 3 oz. thin pus	Abdominal hysterectomy	4 days after operation, "pneumonia"; on sixth day after operation, dyspnea, cyanosis, orthopnea; temperature, 103; pulse rate, 144; respiration, 44	24 hours	
	Case 12: hour-glass stomach with chronic ulcer; small curvature	47	♀	Collapse of left lower lobe, except apex; gastric ulcer	Too ill for operation	"Agonizing gastralgia" on admission; dyspnea; temperature, 101	Death in 2 days	

Case 13: followed "biliary colic—pyemia;" right lower lobe except apex	48	♀	Abscesses of liver (155 oz.); adhesions to diaphragm; right pleura had ½ oz. of bile stained fluid	No operation	?	?
Case 14: followed hydrochloric acid poisoning	30	♀	Esophagus, stomach, duodenum badly damaged; no perforation; general incomplete collapse of both lungs	.....	?	10 hours?
Case 1: diphtheria.....	4	♂	Collapse of right and left lower lobes and portion of right upper lobe; bronchi not described	.....	7 days after "paralysis of the diaphragm"	?
Case 2: diphtheria.....	2½	♂	Patchy areas; collapse of both lungs; slight general dilatation of bronchi which exuded pus on pressure	.....	4 days after paralysis of the diaphragm	?
Case 3: diphtheria.....	3½	♀	Collapse of right lower lobe; scattered areas of collapse in left lung; "some" bronchopneumonia	.....	8 days after paralysis of the diaphragm	?
Case 4: diphtheria.....	4	♀	Collapse of right lower lobe and partial collapse of left lower lobe, emphysema of upper lobes	.....	10 days after paralysis of the diaphragm was noticed	
Case 5: diphtheria.....	5	♀	Collapse of right lower and right upper lobes, except apex; left lower lobe almost entirely collapsed	.....	4 days after paralysis was noticed	
Case 6: diphtheria; "cardiopulmonary crisis"	6	♂	Collapse ?; not seen..	.....	"Well marked signs of asphyxia"; no previous signs of respiratory paralysis	
Case 7: diphtheria.....	4	♂	Small patches of collapse in posterior of both lungs; upper lobes, emphysema	.....	.....	Death 2 days after paralysis of diaphragm
Case 8: diphtheria.....	2½	♂	Collapse of entire posterior of right lobe and left posterior lobe and almost all of right lower lobe	.....	.....	Death 7 days after paralysis
Case 9: diphtheria.....	4	♂	.....	.....	17 days after paralysis of the ribs was noticed; dyspnea; temperature, 102	28 days

* Fifty additional cases are reported by E. A. Mastics, F. A. Spittler and E. P. McNamee: Arch. Surg. **15**: 155 (Aug.) 1927.
† In this table ♂ indicates male; ♀, female.

Statistics—Continued

Writer	Complication or Forerunner; Location of Collapse	Age	Sex†	Deaths and Conditions Found Postmortem	Operation; Anesthetic	Onset; Symptoms	Duration	Treatment
Pasteur, W.: Lancet **2**: 1080, 1910 "Middlesex report"	Case 1: right lower lobe	23	♂	.....	Left inguinal herniotomy; gas-ether chloroform	24 hours after operation; dyspnea, pain in right side of chest; cyanosis; dextrocardia; temperature, 103	16 days	Oxygen
	Case 2: right lower lobe	37	♂	.....	Drainage of right inguinal abscess, very painful; anesthetic ?	4 days after operation; pain and "tightness" in lower right side of chest	11 days	
	Case 3: left lower lobe..	24	♀	.....	Left pyosalpingectomy followed by much pain; anesthetic ?	4 days after operation; pain in left chest; cyanosis; dyspnea; gradual onset; temperature, 103; pulse rate, 120; respiration, 40	8 days expectoration of small amounts yellow viscid sputum	
	Case 4: right middle and lower lobes	45	♀	.....	Cholecystectomy, gas-ether-chloroform	4 days after operation; cyanosis, dyspnea; temperature, 102; pulse rate, 140; respiration, 40	After fifth day; coughed up 2 ounces thick green sputum 24 hours previously	Brandy
	Case 5: chronic cardiac disease; right lower lobe	49	♀	.....	Cholecystostomy .	2 days after operation; recurrent attacks of heart failure; cyanosis, dyspnea; green thick sputum	7 days	Brandy, strychnine, oxygen
Pasteur, W.: Brit. J. Surg. **1**: 587, 1913-1914	Case 1: diphtheria; right lung	..	♂	.....	.....	Temperature, 100; pulse rate, 144; cyanosis, dyspnea, cough	5 days	
	Case 2: diphtheria; both upper lobes	6	♀	.....	.....	4 weeks after diphtheria; cough, dyspnea	28 days?	
	Case 3: right lung.....	23	♂	.....	Left inguinal herniotomy; gas-ether-chloroform	24 hours after operation; dyspnea, cyanosis; temperature, 102; pulse rate, 130	12 hours	
	Case 4: left lower lobe	..	♀	.....	Left pyosalpingectomy; anesthetic ?	72 hours after operation; dyspnea, cyanosis; temperature, 103; pulse rate, 120; respiration, 40; mucopurulent sputum	4 days	
	Case 5: chronic bronchitis preceding; right lung	..	♀	.....	Subtotal hysterectomy; chloroform	48 hours after operation; pain in right side of chest; temperature, 102.6; pulse rate, 120; respiration, 26-32	3 days	
	Case 6: right lower lobe	45	♀	.....	Cholecystectomy; gas-ether-chloroform	4 days after operation; dyspnea, cyanosis; pulse rate, 120; respiration, 40	24 hours	
	Case 7: right lung.....	12	♂	.....	Appendectomy; gas-ether; much mucus	48 hours after operation; slight cough	48 hours	
	Case 8: Left lung.....	23	♀	.....	Acute appendectomy	48 hours after operation; pulse rate, 130; respiration, 24-30	36 hours	

Tidy, H. L., and Phillips, E.: Lancet 1: 1245, 1914	No previous operation nor injury; right lower lobe	29	♂	.....		Sudden pain in right side of chest, dyspnea, cyanosis, sweating, vomiting	41 days	
Elliot and Dingley:# Lancet 1: 306, 1914	Case 1: right lower lobe	31	♂	.....	Double inguinal herniotomy; chloroform	5? days after operation; cyanosis, pain in sternum; mucopurulent sputum; temperature, 101; pulse rate, 144; respiration, 64	9 days	Oxygen
	Case 2: right lower lobe	23	♂	.....	Gangrenous appendectomy; stovaine-spinal anesthesia	46 hours after operation; dyspnea; temperature, 102; pulse rate, 116; respiration, 40	4 days, mucopurulent sputum	
Crymble, P. T.: Brit. J. Surg. 5: 363, 1917-1918								
	Case 6: perforating wound right thorax; right pyopneumothorax; left middle and upper lobes	..	♂	.....	Operation followed recovery from collapse	48 hours; respiration, 40....	6-7 days?	
	Case 9: perforating wound in right side of chest, possibly liver and diaphragm; complete collapse of left lung	..	♂	.....	.....	3 (?) days after injury.....	14 days	
	Case 10: superficial wound in left side of chest; collapse of right lower lobe	..	♂	.....	.....	11 days after injury.....	13 days+	
	Case 11: superficial wound in left side of chest; complete collapse of left lung	..	♂	.....	.....	3 days	16 days	
	Case 12 perforating wound in left upper part of chest; collapse of right upper lobe	..	♂	.....	.....	In "a few days" after injury; retraction of superior mediastinum to right	14 days	
	All, right collapse, of which 6 contralateral and 4 homolateral to wound; 10 cases mentioned, not described							
Rose-Bradford: Quart. J. Med. 12: 127, 1918-1919	Superficial wound in the skin, face, right side of the back, one deep wound at angle of scapula; almost complete collapse of left lung	41	♂	.....	.....	2 days after wound; no subjective signs, slight cough 3 days after onset	15 days	
	Penetrating wound in iliac crest; collapse of left lower lobe	22	♂	.....	.....	6 days after injury; mucoid cough	14 days ?	

Nine more cases mentioned.
† In this table ♂ indicates male; ♀, female.

Statistics—Continued

Writer	Complication or Forerunner; Location of Collapse	Age	Sex†	Deaths and Conditions Found Postmortem	Operation; Anesthetic	Onset; Symptoms	Duration	Treatment
Scholtz, C. J.: M. J. S. Africa **16**: 202, 1921	Right lower lobe.....	37	♂	.....	Left inguinal herniotomy; nitrous-oxide-ether	2½ hours; pain, cyanosis....	8 days	
Eccles, M.: W. Lond. M. J. vol. 16	.....	..	..	.....	Lumbar nephrectomy			
St. Bartholomew Hosp. J. 1914	Injury to rectum by iron spike	9	♂	.....	Repair of rectum	24 hours	24 hours	
Scrimger, F. A. C.: Surg. Gynec. Obst. **32**: 486, 1921	Case 1: right lower lobe	34	♂	.....	Left inguinal herniotomy	24 hours; orthopnea, cough	15 days	
	Case 2: left lower lobe	..	♂	.....	Chronic appendectomy	3 days; dyspnea; temperature, 102.2; pulse rate, 120; respiration, 30	24 hours after coughing up blood stained mucus, recovery	
	Case 3: left lower lobe	..	♂	.....	Strangulated right inguinal herniotomy	24 hours; pain, dyspnea, temperature, 101; pulse rate, 120; respiration, 36	Recovery 24 hours after sudden coughing and drawing long breath	
	Case 4: right complete	..	♂	.....	Right inguinal herniotomy	13 days; temperature, 101; pulse rate, 108; respiration, 28; no distress	4 days	Blowing exercises
	Case 5: coughing before operation; right lower lobe; complete collapse of lung 24 hours later	..	♂	.....	Right inguinal herniotomy	24 hours; temperature, 100; pulse rate, 112; respiration, 36	27 days?	Blowing exercises
	Case 6: right lower lobe	..	♂	.....	Subacute appendectomy	48 hours; temperature, 101; pulse rate, 120; respiration, 40; dyspnea, cough	9 days	Blowing exercises
	Case 7: left lower lobe	..	♂	.....	Hemorrhoidectomy	24 hours; cough, mucus; temperature, 100; respiration, 28	10 days	
Elwyn, H.: J. A. M. A. **39**: 2154, 1922	Case 12: right and left lower lobes	..	♀	Collapse of lower part of both lower lobes	Panhysterectomy; salpingo-oophorectomy	48 hours	6 days	
	Case 13: right and left lower lobes	..	♀	Collapse of right and left lower lobes	Hysterectomy; bilateral salpingo-oophorectomy; nitrous oxide-ether	24 hours; diminished breathing; cough	5 days	
	Case 14: streptococcus bacteremia following operation; right lower lobe	..	♀	Collapse of right lower lobe	Left axillary cellulitis; nitrous-oxide-oxygen	Same day?	2 days	
	Case 15: left lower lobe	..	..	.....	Cholecystectomy..	24 hours; rise in temperature	48 hours	
Elwyn-Girsdansky: J. A. M. A. **79**: 718, 1922	Complete collapse of right lung	14	♂	.....	For stab wound in the epigastrium to left midline	24 hours; cough, mucopurulent sputum; temperature, 101; dyspnea; cyanosis	13 days	

Thoenes: Monatschr. f. Kinderh. **22**: 333, 1922	Complicating pneumonia (11 cases)	17 days to 13 mo.	..	.....				
Scrimger, F. A. C.: Am. J. Surg. **36**: 50, 1922	Case 8: right lower lobe	30	♂	.....	Gangrenous; appendectomy; nitrous-oxide-ether	20 hours after operation; cough, thick, blood stained sputum; pain in chest; temperature, 102; pulse rate, 120; respiration, 30	4 days	
	Case 9: right and left base	28	♂	.....	Left inguinal herniotomy; nitrous-oxide-ether	10 hours after operation; cough, dyspnea, cyanosis, frothy expectoration; no dextrocardia; temperature, 102; pulse rate, 160; respiration, 40	4 days	
	Case 10: right base.....	21	♂	.....	Gangrenous; appendectomy; gas-ether	15 hours after operation; cough, dyspnea, expectoration; temperature, 100; pulse rate, 160; respiration, 30	4 days	
	Case 11: asthmatic patient; collapse of left base complicated by pneumonia, 4th day	13	♂	....	Gangrenous appendectomy; local anesthesia, gas-oxygen	12 hours after operation; pain in left chest, orthopnea, dyspnea, cough; temperature, 104; pulse rate, 140; respiration, 40	3 days, temp. 100, pulse 120, resp. 30; fourth day, temp. 102 = pneumonia; well 9 days later	Atropine
Hirschbroeck, F. J.: Am. J. M. Sc. **164**: 268, 1922	Case 1: left lower lobe	38	♀	.....	Ovarian cyst; appendectomy; laparotomy	48 hours; temperature, 101; pulse rate, 130	4 days?	
	Case 2: right lower lobe	49	♂	.....	Acute appendectomy	24 hours; temperature, 102; pulse rate 110; dyspnea	3 days	
	Case 3: right and left lower lobes	48	♂	Collapse of right and left lower lobes; bronchi not described	Appendectomy; posterior gastro-enterostomy	6 days; dyspnea, no displacement of the heart	48 hours	
Elwyn, H.: J. A. M. A. **82**: 384, 1924	Case 1: right lower lobe	32	♂	.....	Left indirect inguinal herniotomy; gas-ether	24 hours; cough, pain.....	14 days	
	Case 2: right lower lobe	35	♂	.....	Colectomy for carcinoma of the ileocecal junction; gas-oxygen	24 hours; temperature, 102..	8 days ++	
Lee, W. E.: Ann. Surg. **79**: 518, 1924	Right lower lobe, complicated by purulent pneumonia in left upper lobe	..	♂	Definite plug of mucus in right lower bronchus	Strangulated femoral hernia (side); local anesthesia	72 hours	7 days	
Leopold, S. S.: Am. J. M. Sc. **167**: 421, 1924	Case 1: complete collapse of right lung	27	♂	.....	Ventral hernia; ether	24 hours; sudden; dyspnea, cyanosis; increase in temperature, pulse rate and respiration	5 days ++	
	Case 2: complete collapse of right lung	22	♂	.....	Left inguinal herniotomy; ether, 6 ounces	3 days, sudden; dyspnea, cyanosis; increase in temperature, pulse rate and respiration	5 days ?	
	Case 3: complete collapse of right lung	25	♂	.....	Right inguinal herniotomy; ether, 8 ounces	2 days, sudden; dyspnea, cyanosis; increase in temperature, pulse rate and respiration	5 days?; recurrent clearing and collapse	
	Case 4: left lower lobe	24	♂	.....	Left inguinal herniotomy	24 hours, sudden; symptoms as above	2 days?	

† In this table ♂ indicates male; ♀, female.

Statistics—Continued

Writer	Complication or Forerunner; Location of Collapse	Age	Sex†	Deaths and Conditions Found Postmortem	Operation; Anesthetic	Onset; Symptoms	Duration	Treatment
Ritvo, M.: Am. J. Roentgenol. **11**: 337, 1924	Collapse of right lung	28	♂	.....	Relief of postoperative appendical adhesions; gas-ether	24 hours after operation: dyspnea, "tightness" of the chest; thin yellow sputum; temperature, 102; pulse rate, 105; respiration, 30	Improved 6th day, recovered 8th day after operation	
Holmes, G. W.: Am. J. Roentgenol. **11**: 509, 1924	Case 1: collapse of right lower lobe ?	20	♀	.....	Left oophorectomy; right salpingo-oophorectomy; appendectomy; gas-ether; excessive mucus	36 hours after operation; dyspnea; rise in temperature, pulse rate and respiration; small amount of purulent sputum	24 hours	
	Case 2: complete collapse of right lung	14	♂	.....	Left indirect inguinal hernia; procain hydrochloride	48 hours after operation; dyspnea; dry cough; pain in right side of chest	8 days	
	Case 3: chronic "smokers' cough;" right lower lobe	19	♂	.....	Subacute appendectomy; ether; excessive ? mucus	48 hours after operation: chill, cough, dyspnea, cyanosis; temperature, 103; respiration, 48	8 days+	
	Case 4: acute upper respiratory infection 10 days before admission; complete collapse of right lung	40	♂	....	Bilateral herniotomy; epidural anesthesia	1½ hours after operation; cough, purulent sputum, no pain, no dyspnea; temperature, 102	"Several days" after operation	
Abt, I. A.: Am. J. Dis. Child. **30**: 347, 1925	Right lower lobe; collapse complicated by pneumonia; right upper lobe ? on fourth day of collapse	10	♂	.....	Appendectomy for gangrenous appendix; ether	48 hours after operation; dyspnea; cyanosis; painful cough; right side of chest immobile; interspaces widened	12 days	Dyspnea relieved by 1/12 grain morphine
Lee, W. E.: Bull. Ayer Clin. Lab. 9, 1925	Case 1: had bronchitis on admission; collapse of right lung	14	♂	.....	For perforated appendix with abscess; gas-oxygen	5 days after operation; cough; thick green sputum; cyanosis; temperature rose 5 degrees; pulse rate, 152; respiration, 52; pain in right side of chest; right side of chest retracted	6 days, during which increasing quantities of thick green sputum	
	Case 2: bronchitis and cough on admission; pregnancy; collapse of right and left lower lobes	18	♀	Local peritonitis; diaphragm right third space, left fifth space; heart drawn to left; lobular collapse of lower part of right and left lower lobes; main bronchi and bronchioles filled with thick mucopus	Cesarean section..	3 days after operation.....	2 days	

	Case 3: collapse of right lower lobe; lobular collapse of right middle lobe	24	♀	General peritonitis; thick mucopus in right middle and lower bronchi; collapse of lower third of lower lobe; scattered areas on posterior of right middle and lower lobes	Appendectomy (peritonitis)	?	Death 6 days after operation
	Case 4: carcinoma of the head and pancreas; collapse of both lower lobes	58	♂	Carcinoma of head and pancreas; liver large (metastases); right diaphragm high and tense; mucus in bronchi not excessive	....	?	?
	Carcinoma of the esophagus; collapse of right lower lobe	47	♂	Carcinoma of esophagus just above cardia; metastases to liver (3× normal); right diaphragm high and tense; right lower bronchus had a moderate amount of thick bloody mucus	.....	?; with rise in temperature and signs; right lower bronchopneumonia	Death within 24 hours
Jackson, C., and Lee, W. E.: Ann. Surg. **82**: 364, 1925	Case 1: collapse of right lung	14	♂	.....	Gangrenous appendectomy; localized abscess; gas-oxygen	5 days after operation; cough; thick green sputum for 5 days; temperature, 105; pulse rate, 152; respiration, 52	42 days
	Case 2: partial collapse of right base and left base	18	♀	Collapse of left base and partial of right base; local peritonitis; paralytic ileus; hydro-ureter; hydronephrosis; thick mucopus in left lower bronchus	Cesarean section..	3 days	6 hours; death
Scott, W. J. M.: Arch. Surg. **10**: 73, 1925	Case 37: collapse of right lung	18	♂	.....	Appendectomy; upper exploratory; ether, (cough and mucus during induction)	Cough 2 hours after operation; 13 hours pain in right side of chest; dyspnea, not toxic; temperature, 102.4; pulse rate, 120; respiration, 40	48 hours, recovery starts; 7 days, recovery complete
	Case 38: collapse of right lung	20	♂	.....	Appendectomy; repair ventral hernia; ether	24 hours after operation middle lobe of right lung collapsed; 48 hours, total right lung collapsed; temperature, 101; pulse rate, 120; respiration, 52	16 days
	Case 39: chronic bronchitis, 4 years; collapse of right lung	19	♂	.....	Appendectomy; gas, ether	48 hours	7 days; 4 days later coughed up 120 cc. of blood
	Case 40: collapse of left lung	26	♂	.....	Appendectomy; ether	24 hours after operation; dyspnea; pain in the chest; cough; thick, tenacious, mucopurulent sputum	14 days+

† In this table ♂ indicates male; ♀, female.

Statistics—Continued

Writer	Complication or Forerunner; Location of Collapse	Age	Sex†	Deaths and Conditions Found Postmortem	Operation; Anesthetic	Onset; Symptoms	Duration	Treatment
Griffith, T. P. C.: M. J. & Record **123**: 103, 1926	Preceded by diarrhea, vomiting and nasal regurgitation; collapse of right lung	23 days (premature)	♀	.....	.....	Slight cough and duskiness	7 days	
Gwyn, N. B.: Internat. Clin. 136, 1926	Case 1: followed pneumonia of right lower lobe; collapse of right upper lobe	60	♀	.....	.....	Fifth day of pneumonia; dyspnea, cyanosis, dextrocardia	3-4 days	
	Case 2: followed pneumonia of left lower lobe; collapse of left lower lobe	..	..	.....	.....	?; roentgen ray confirmed; heart shifted to left	7 days	
	Case 3: pneumonia of left base; collapse of left upper lobe	6	♂	.....	.....	?; no distress.....	3 days	
	Case 4: followed pericarditis with effusion; complete collapse of left lung	63	♀	.....	.....	Sudden; dyspnea; heart shifted to left	Gradual clearing	
	Case 5: associated with lobular pneumonia; left upper and middle lobes	..	..	Lobular pneumonia of the whole left lung and lower loft lobe				
Gammons, H. F.: Boston M. & S. J. **194**: 538, 1926	Tuberculosis of left upper lobe preceded by collapse of right upper lobe	30	♂	.....	.....	Gradual; coughing; 24 ounces purulent sputum; hemoptysis; increase in temperature and respiration; pain over right apex	Markedly improved in 6 days	500 cc. of air injected into right pleural cavity; 2 days, 3 times a day
Rigler, L. G.: Minnesota Med. **9**: 326, 1926	Case 1: complete collapse of right lung	18	♂	.....	Appendectomy; ether	48 hours after operation; dyspnea, cyanosis, cough; temperature, 102	14 days	
	Case 2	?	..	.....	Cholecystectomy; local anesthesia	60 hours; cyanosis; temperature, 103	4 days	
	Case 3	10	..	.....	Appendectomy			
	Case 4: right complete followed by right empyema	..	♀	.....	Salpingectomy, bilateral; ether	24 hours after operation; temperature, 100.2; cough, cyanosis; heart and mediastinum to the right; after right empyema the heart shifted to the left	13 days	
	Case 5: collapse complicated by pneumonia in right upper lobe; collapse of right middle and lower lobes	..	..	.....	Salpingectomy, bilateral	Onset ?; pneumonia ? third day, rusty sputum		
	Case 6: followed fracture of the pelvis	..	♂	.....	.....	12 hours after injury; right middle and lower lobes	6 days+; large amount mucoid sputum	
	Case 7‡ } mentioned Case 8‡ } not described							

Mason, R. L.: Surg. Clin. N. Amer. **6**:739, 1926	Collapse of left lung...	18	♂	.....	Bilateral inguinal hernia; gas-ether	55 hours after operation; dyspnea; pain beneath xyphoid; cough; cyanosis; temperature, 102; pulse rate, 120; respiration, 48	72 hours	
Singer, J. J., cited by Mason, R. L.: Surg. Clin. N. Amer. **6**:739, 1926	.....	..	..	.....	Appendectomy ...	.....	.....	Rolling from side to side
Herrman, W. G., cited by Mason, R. L.: Surg. Clin. N. Amer. **6**:739, 1926	Associated with pneumothorax	..	..	.....	.....	.....	.....	Rolling from side to side
Kletz, N.: Lancet **212**: 179, 1927	Followed acute meningitis; collapse of right and left lower lobes	18	♂	Collapse of right and left lobes; lungs leathery; few ounces of fluid in both pleura; meningococci and streptococci in cerebral sulci	.....	3 days after meningitis; dyspnea, "gripping" of chest; temperature, 101.4; pulse rate, 126; collapse of left lung 24 hours after right lung collapsed	24 hours	
Harrington, S. W., et al.: Ann. Surg. **35**: 152, 1927	Collapse of left lung...	28	♂	.....	For right hydronephrosis; ether	24 hours; "burning" under sternum; pain in left side of chest; temperature, 102; pulse rate, 110; respiration, 28	15 minutes	Bronchoscopy; aspiration, 300 cc. thin serous secretion
Sante, L. R.: J. A. M. A. **88**: 1539, 1927	Case 1: injury of left hip; collapse of right lung	16	♂	.....	.....	24 hours after injury.....	Cleared up 3 minutes after rolling from left side to back (fluoroscopy)	Rolling from side to back (accidental)
	Case 2: collapse of left lung	30	♂	.....	Kidney fixation (side?); ethylene-ether	7 days after operation temperature, 102.8; pain in chest	7 days	Rolling from side to side, plus cough
	Case 3: gunshot wound in abdomen, pelvis and left wrist; collapse of right lung	23	♂	.....	Exploratory laparotomy; drainage of subcutaneous tissues	4 days after operation; dyspnea, pain in right side of chest and at umbilicus; temperature, 102; respiration, 40	9 days; cleared in several minutes after treatment	As above; coughed up 5 cc. of mucus (globular, gray, "gumdrop")
Bergamini, H., and Shepard, L. A.: Ann. Surg. **86**: 35 (July) 1927	Case 1, M. L.: collapse of left lung	44	♀	Collapse of one lung; edema of glottis; status lymphaticus; bronchi clear	Occurred near end of operation under gas-oxygen-ether; hysterectomy; salpingo-oophorectomy	Patient stopped breathing near end of operation	.....	Artificial respiration; stimulants
	Case 2	69	♂	Collapse of both lungs	Laparotomy; gas-oxygen-ether	Sudden, at end of operation	.....	Artificial respiration; stimulants
Hearn, W. P., and Clerf, L. H.: Ann. Surg. **85**: 54, 1927	Collapse of left lower lobe	8	..	.....	Closure of gastrostomy fistula; cough, much mucus during anesthesia	36 hours after operation; temperature, 103.4; diagnosis made 64 hours after operation	23 days+; 6 aspirations done, less secretion each time	Bronchoscopy 8 hrs. after diagnosis made; aspiration of thick, tenacious, yellow sputum from trachea and left bronchus

† In this table ♂ indicates male; ♀, female.
‡ Fracture of pelvis; onset 12 hours; duration 3 days.
§ Fracture of spine and right ribs; recovery complete; cleared up shortly.

SUMMARY AND CONCLUSIONS

1. The determining cause of massive atelectasis of the lung (or patchy atelectasis), whether it is postoperative, spontaneous or secondary to obstruction by a foreign body or to infection, producing bronchial exudate of membranes, is always a complete obstruction of a bronchus (main, secondary or small) or compression of the lung (by effusion, pneumothorax, etc.) which abolishes the effects of the intrapleural negative pressure.

2. The term "collapse" may lead to confusion because it does not always signify "airlessness," which is meant when the term "atelectasis" is used. Apneumatosis would be an even more adequate term than atelectasis.

3. The obstruction of a bronchus in postoperative cases is due to a plugging by mucus, which may be present at autopsy or which may have been expelled before death. It is possible that "plugging of a bronchus" may be effected by thin secretion or exudation occluding the lumen of a bronchus, so that the word "plug" must probably not be taken too literally. This interesting point is being given further study.

4. As an aid to the accumulation of mucus, there are all the causes or factors which diminish respiratory movements and cough, namely, pain, paralysis of the respiratory muscles (intercostal, diaphragm) reflex respiratory inhibition from whatever cause, general cachexia, recumbency or narcotics.

5. Spastic contraction of the bronchial muscle does not produce apneumatosis, but it does produce emphysema.

6. The existence of so-called "angioneurotic edema" of the lung has never been proved, clinically or experimentally. In the cases of acute anaphylactic shock in the guinea-pig, emphysema and not "collapse" is produced even when edema occurs (Hoover).

7. Atelectasis may predispose to infection by fixation in the parenchyma of the lung of septic emboli or microbes present in the circulating blood. This suggests the usefulness of the investigation of lobar pneumonia in possible relation to obstructive atelectasis because this could explain the anatomic lobar disposition of the disease in relation to the obstruction of a bronchus by: (1) the pneumonic sputum which is so viscid and tenacious and (2) the diminished force of expectoration.

8. From the foregoing considerations a definite therapeutic conclusion may be drawn. When simple methods, such as shaking or changing the position of the patient, are not quickly successful, the obstructing agent must be removed by bronchoscopy with aspiration or extraction in order to hasten recovery and to avoid further septic complications, such as bronchopneumonia, pneumonia or abscess. Artificial pneumothorax, as advocated by Elliot and Dingley, may be a palliative but is not a therapeutic measure.

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KÖHLER'S DISEASE *

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Köhler's disease is a peculiar condition in which the tarsal navicular bone is abnormal in shape, size and density. It was first described by Köhler of Wiesbaden in 1908. It affects boys more often than girls in the proportion of about two to one. The age limits are from 1 to 10 years, the great majority of cases occurring in patients between the ages of 3 and 7. The disease is characterized by pain which is made worse by exercise. Sensitiveness is also present over the navicular bone, possibly accompanied by redness and swelling. The patient limps, and may walk on the lateral border of his foot. The symptoms persist for a longer or shorter time, and then disappear. Complete recovery always takes place with any treatment.

The diagnosis is made with the roentgen rays. The bone nucleus of the navicular bone is usually narrow, that is, the distance from the proximal to the distal border is diminished, but the gap between it and the talus and that between it and the cuneiform bone indicate the presence of cartilage. The bone itself is usually irregular in shape, and irregular and dense in structure, so dense that the usual architecture cannot be outlined. On the other hand, the bone nucleus is seen occasionally as a faint irregular area, much smaller than that on the other foot.

The cause of Köhler's disease has never been established. Operations have been rare; one or two surgeons have reported positive cultures, but their reports are not conclusive. The condition has been ascribed to injury, fracture and obscure infection. Some recent writers consider the condition similar to Legg's disease and to Osgood-Schlatter's disease. In my book, "Inflammation in Bones and Joints," published in 1923, I classified the disease with these and with the true joint mouse found in adolescence in the medial condyle of the femur, and ascribed them all to an error in the development of the bone nucleus. Each condition occurs in boys predominately, at a certain age, and has a characteristic roentgen-ray appearance.

REPORT OF CASES

CASE 1.—A boy, aged 4½ years, complained of pain in his right foot. He limped at times. The mother noticed that he turned his foot out when he walked, but she had never noticed any swelling. The foot was in slight eversion. All motions were free. A sensitive prominence was present over the navicular bone. Roentgen-ray examinations showed a dense, irregular navicular bone, "much

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compressed from before backward." The other navicular bone was normal. The patient did not return to the clinic. Four and one-half years later, he was persuaded to return for roentgen-ray examination. He had not had much trouble since the previous visit, and had recovered completely. No sign of the disease was present, and there was no deformity. The roentgenograms showed normal navicular bones.

Fig. 1 (case 1).—Lateral view of foot. Note the density of the bone, the irregularity of its structure, the comparative smoothness of its proximal and distal borders and the clear space between it and the cuboid and talus. In a destructive disease of the bone, its structure would be rarefied, and its collapse would mean the approximation of the cuboid to the talus.

Fig. 2.—The same foot as shown in figure 1, four and one-half years later showing restoration of function and structure without treatment.

CASE 2.—A boy, aged 6½ years, had had moderate pain in his left foot for five or six days. He walked with a limp, and the foot had been swollen. The swelling had decreased and the pain had disappeared. Examination showed only a "typical case of Köhler's disease," whereby I am led to infer that the swelling and sensitiveness were present over the navicular. The roentgen ray showed that

the navicular was "compressed and markedly irregular." One week later, the boy returned to the clinic. He walked without a limp. His left navicular bone was slightly prominent, but it was not sensitive on pressure. There are two other children in the family who are alive and well. The patient had an indication of an old Harrison's groove and fairly marked knock knees, but no other signs of rickets. Almost four years later, he was bribed to return to the clinic

Fig. 3 (case 2).—Similar to figure 1, but with slightly greater irregularity of contour.

Fig. 4 (case 2).—Complete recovery symptomatically and structurally after almost four years, without treatment.

for roentgen-ray examinations. He had not had any further trouble with his foot. Clinically, there was no sign of disease and no deformity except, perhaps, a minimal prominence of the navicular bone. Roentgenograms showed two normal navicular bones.

CASE 3.—A boy, aged 6½ years, was brought to the clinic with a history of swelling in his left foot for two weeks. Little pain was present at first, but it

had increased for the last three days before he came to the clinic. At first walking was difficult, but for the past three days it had been impossible. Pain was much worse when the patient attempted to use his foot. Examination showed swelling of the left foot, with increase in local temperature and tenderness over the navicular and the cuboid bones. The roentgen ray showed the characteristic changes of Köhler's disease. "The changes were not essentially different in one

Fig. 5 (case 3).—Left foot, showing density and irregularity of the navicular bone. This was the painful foot.

Fig. 6 (case 3).—The right foot which was the foot without symptoms, The navicular bone is more abnormal than in the foot of which the patient complained, and appears to be developing from two nuclei.

foot from those in the other. The left scaphoid outlines showed definitely that the scaphoid bone had collapsed." Anteroposterior roentgenograms showed also irregularity of development of the medial cuneiform and fifth metatarsal bones. The patient was sent to the hospital, and the foot was wrapped in cotton. Salicylates were administered. The pain at first was intense. After four days the foot was strapped, and the patient was discharged. He did not have any

Fig. 7 (case 3).—The left foot, shown in figure 5, three years later. Complete recovery without treatment.

Fig. 8 (case 3).—The right foot shown in figure 6, three years later. Complete recovery, without treatment.

Fig. 9 (case 4).—The navicular bone shows a rather dense cortex, similar to that in figure 1. Its smooth surface is not that of a diseased bone.

pain in the foot afterward. By request, he was brought to the clinic for examination three years later. He had the slight prominence of the navicular region seen so often. The roentgenograms showed an almost normal appearance. The navicular bones had developed well and almost symmetrically. The left one was a millimeter thinner at the center and 1.5 mm. broader from above below than the right.

Fig. 10 (case 4).—The navicular bone three years after first roentgenogram. Complete restoration of function and structure after almost three years, practically without treatment.

Fig. 11 (case 5).—Note swelling in the soft parts. This navicular bone has not the same density as the others. It was supposed to be "collapsed," but if it had been, the talus and cuboid would be approximated. On the contrary, the normal distance is present between them.

CASE 4.—A boy, aged 5 years, three weeks before he was brought to the clinic, awoke during the night complaining of pain in his left foot; the foot was swollen. It remained swollen for two days, and then became entirely normal. Further trouble did not occur until four days ago, when the boy again awoke during the night with pain and swelling in the same foot. The pain gradually decreased. There was no history of injury. Examination showed slight swelling

in the region of the left navicular bone, with sensitiveness. Extremes of motion were painful. The roentgen rays showed the typical appearance of Köhler's disease. The foot was strapped with adhesive tape, and all symptoms had disappeared in about a month. Thirteen months later, the boy was again brought to the clinic complaining of pain in the right foot. The roentgenogram of the right

Fig. 12 (case 5).—Roentgenogram taken four months after figure 11. The disease seems to have progressed, but the condition was said to have disappeared shortly afterward.

Fig. 13 (case 6).—In this case there is simply marked irregularity of structure of the diminutive navicular bone, without the usual increase in density of Köhler's disease.

foot did not show any abnormalities, that of the left showed a solidification of the left navicular bone as compared with a roentgenogram taken the previous year, but some irregularity in structure was still present, although all symptoms were absent. Eighteen months later, or almost three years after the first visit,

the boy was again brought to the clinic. He then had had pain in the left foot for one day. With the exception of slight tenderness over the navicular bone, the results of the examination were negative. Roentgenograms of both feet did not show any abnormalities. The foot was strapped, and in one week was perfectly normal.

CASE 5.—A boy, aged 4 years, had complained of pain in his right foot and a limp of two months' duration. He had been healthy previously, except for a little bronchial trouble, and pneumonia eight months before. The personal and family histories were not significant. About two months previously, when he arose one morning he complained of pain in his right foot, and limped. The pain grew worse for one week, and the foot swelled, but then the condition improved. For the past week it had grown worse again. The boy was in good physical condition, but he limped badly. The right foot was in slight abduction. Swelling, sensitiveness, redness and increased local temperature of the tarsus were present, with limitation of abduction and adduction and muscular spasm. The sensitiveness was most marked about the talonavicular joint. My tentative diagnosis was Köhler's disease, but the members of the department of radiology were inclined to favor the diagnosis of tuberculosis. The consultant agreed with this opinion. The foot was therefore put in plaster of paris. Four months later, other roentgenograms showed less of the shadow of the navicular bone than the first, and the roentgenographer reported: "The observed progress of the disease makes tuberculosis much the most likely diagnosis." Two or three months later, the parents are said to have removed the plaster of paris and to have found the foot normal. The father expressed himself as indignant that he had been put to the expense and the trouble of treatment. Roentgenograms of this patient could not be obtained.

CASE 6.—A girl, aged 5 years, was said to have jumped and sprained her right foot about one year previously. The foot had swelled at intervals ever since. It had not been painful at any time, and the child had never limped. At the time of examination, no swelling, deformity nor redness was present, nor was there any evidence of injury or disease. The roentgen rays showed a rudimentary navicular bone. The foot was strapped, and the condition soon improved. Further examination or check-up with the roentgen rays could not be obtained, but three years later, the family physician reported that the foot had been perfectly normal.

SUMMARY

Six cases, those of five boys and one girl, ranging in age between 4 and 6½ years, have been reported. In three, the right foot was affected; in two, the left. In one case, the symptoms were present in the left foot only, but the roentgen rays showed the appearance of the condition in both feet. In all but one case, the roentgenogram was so characteristic that the diagnosis could be made without difficulty; in that case, the diagnosis was uncertain, and the condition cleared up spontaneously. There was correspondence between the clinical picture and the roentgen-ray observations only in the fifth case. The clinical picture was that of a mild injury or infection; the roentgen-ray picture, that of great "damage" to the bone. The finding of marked abnormality, by roentgen rays in an apparently normal foot shows that the "disease" may

exist without causing any symptoms or physical signs. A close scrutiny of the roentgenograms often reveals greater or less abnormalities in the other bones of the feet. In spite of the great abnormality in structure, as shown by the roentgen rays, five patients recovered promptly with little or no treatment. The patient in case 5 wore a plaster for about six months, and then recovered. Not only does the patient recover promptly, but roentgen-ray examination shows that the apparently hopelessly damaged bone slowly develops and becomes normal in structure and in shape.

CONCLUSION

Köhler's disease is a developmental anomaly of the tarsal navicular bone accompanied by symptoms. Patients with this disease always recover spontaneously. As in Osgood-Schlatter's and in Legg's disease, the anomaly may exist without symptoms. The patient should be treated symptomatically.

TORSION OF THE OMENTUM WITHOUT HERNIA

REPORT OF TWO CASES

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CLASSIFICATION OF TORSION OF THE OMENTUM

The various types of torsion of the omentum have been classified as follows:

1. Torsion of the omentum unassociated with hernia, adhesions or tumors.
2. Torsion without hernia, but with old adhesions at one or more points.
3. Torsion in a hernial sac or in the abdomen, intimately associated with the hernia.
4. Torsion in the abdomen in conjunction with an existing or preexisting hernia, but having no connection with it.
5. Torsion without hernia, associated with tumors of the omentum.
6. Torsion without hernia, associated with tumors in the abdomen or pregnancy or otherwise complicated.

Pure torsion of the omentum (group 1) unassociated with hernia, adhesions or tumors, is a rare condition. A search of the literature has revealed only twenty-four cases (8, 10, 14, 15, 17, 21, 22, 24, 25, 28, 29, 30, 31, 32, 33, 35, 36, 37, 39, 40, 41 and 42) to which my two cases are added.

CASE 1.—*History.*—A. R., a man, aged 55, weighing 185 pounds (83.9 Kg.), entered the Presbyterian Hospital on Aug. 7, 1926, with torsion of the omentum and symptoms of acute cholecystitis. He complained of severe sharp pains and tenderness in the right upper quadrant of the abdomen of forty-eight hours' duration. He had fallen across the top of an automobile on his abdomen from the top of a stepladder forty-eight hours previously. He did not notice any pain until about four hours after the fall, when pain started over a small area in the right upper quadrant. It was cramplike and sharp, coming from every five to fifteen minutes and lasting for from three to four minutes. The pain became progressively worse, and the area of tenderness became larger. He said that it felt as though there were a mass pushing forward in this area. He worked only the remainder of the first day, and was unable to get much sleep either night. The pain was increased on breathing or on motion. It did not radiate and was no more marked when the patient was standing than when lying down. Vomiting and nausea were entirely absent. The appetite was good the first and second days, but he was unable to eat the morning of entrance because of severe pain. The bowels moved regularly.

He had not any sickness for forty years. At the age of 14, he had suffered with so-called typhoid pneumonia. There was no history of abdominal disorder. The weight had been the same for several years.

Physicial Examination.—The patient was large and moderately obese. There was no hernia. There was marked rigidity of the upper two thirds of the right side of the abdomen which extended somewhat posteriorly. Tenderness was exquisite over this area. A mass could not be palpated, probably owing to the rigidity, tenderness and obesity. The temperature was 99.6 F.; the pulse rate, 100 and the white blood cell count, 17,000. Otherwise the results of the examination were negative.

Operation.—Ethylene and ether anesthesia was given. An incision was made near the midline in the upper part of the abdomen. There was a quantity of free clear fluid in the peritoneal cavity. A large mass was seen at the right side of the midline, consisting of a large fat omentum. This was discolored bluish and red in areas. There was a complete twist to be seen at its base just below the stomach. The omentum was not attached to the transverse colon. The mass extended across the transverse colon for about 4 inches (10.16 cm.) and lay beneath the hepatic flexure of the colon, where it was partially attached (fig. 1) by recent fibrinous adhesions to the epiploic appendixes. The surrounding tissues were edematous. There was no evident constriction of the transverse colon. The whole omentum was twisted and strangulated with hemorrhagic areas throughout. It was resected above the twisted region, which was somewhat smaller than the thick, fat distal mass. The base was completely twisted one and one-half turns. The appendix, which was bound down and not inflamed, was not removed.

The gallbladder, stomach and pancreas were normal.

Pathologic Examination.—The specimen consisted of a firm mass of omental tissue, 11 cm. wide and 2 cm. thick. The anterior surface was smooth, lobulated, glistening, reddish yellow with darker streaks of red running between each lobulation. About one third of the middle of the posterior surface was lemon yellow. The remainder was like the anterior surface. The cut edge was dark red and contained congested blood vessels, which with the omental tissue surrounding the base had twisted one and one-half times. Surfaces made by cross-cutting had a core of dark brown, distinctly lobulated, slightly softer tissue than the covering. This was surrounded by reddish-yellow, firm tissue 2 mm. thick in the anterior surface, 5 mm. in the posterior and about 15 mm. from the sides.

On one side of the omentum was a small tag or process the pedicle of which had also twisted (fig. 1).

Microscopic examination of a section showed many fat lobules in which the fat cells were separated by blood. The lymphatics were filled with polymorphonuclear leukocytes and a few lymphocytes. In the connective tissue, likewise, were many polymorphonuclears.

Postoperative Course.—There was an acute dilatation of the stomach on the second day, which was severe and lasted three days in spite of repeated aspirations. On the eleventh day, bronchopneumonia developed in the right base, running an atypical course and possibly being embolic in origin. It lasted about six days. The patient was in the hospital for twenty-six days and made a complete recovery.

One year later he was entirely free from complaints and doing his usual work

CASE 2.—*History.*—C. A., a man, aged 49, weighing 152 pounds (68.9 Kg.), entered the Presbyterian Hospital on Oct. 21, 1926, with torsion of the omentum and symptoms of acute appendicitis. He complained of sharp, knifelike pains in the lower right quadrant for two days. The distress in the right lower quadrant began four days previously. It followed the unusual work of putting up storm windows, in which the patient strained upward in lifting and raising the windows. The sharp pains began suddenly two days later as he was getting up from his desk. They recurred on any exertion and gradually became worse. He ate lunch,

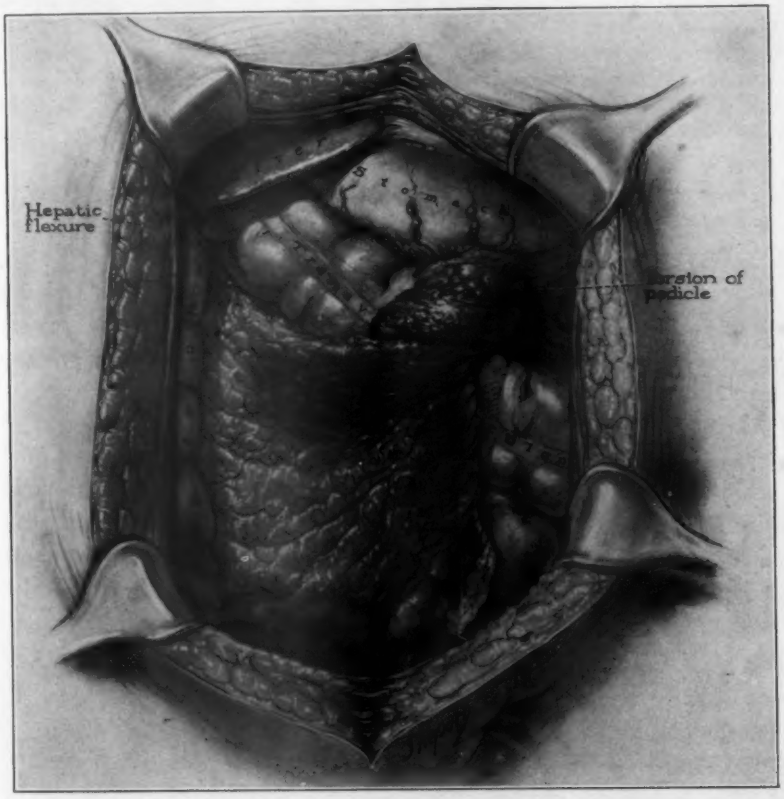

Fig. 1 (case 1).—Torsion with strangulation of the entire obese omentum following a fall.

however, and remained at work that day. The pains were so severe and knifelike on the morning of entrance to the hospital that he could not get up. They remained in the right lower quadrant, extending somewhat around to the side, but did not radiate. He felt as though his back would give way. Vomiting and nausea were absent. His appetite was good until noon on the day of entrance. The bowels moved regularly.

There was no history of previous illness, operation, or abdominal disorder; he had always felt well. His weight had remained the same for several years. His occupation was largely desk work.

Physical Examination.—The patient was moderately obese and looked anxious. There was no hernia. The abdomen was prominent, due to obesity. There was exquisite tenderness over the right lower quadrant. This extended upward, somewhat along the region of the ascending colon and laterally. Muscle spasm was marked but rigidity was not definite. A mass could not be felt, but palpation was not satisfactory.

The temperature was 99.4 F.; the pulse rate, 92; leukocytes, 12,000. Otherwise results of the examination were negative.

Operation.—Ethylene anesthesia was given. A muscle splitting incision was made. The peritoneum was thickened and edematous. There was considerable clear fluid in the peritoneal cavity. The appendix, which was congested and felt to contain a fecalith, was removed. The surrounding structures were also con-

Fig. 2 (case 2).—Strangulation of the distal half of the obese omentum by torsion following unusual physical exertion. The narrow pedicle is apparently of long standing.

gested. The changes in the appendix were not thought sufficient to explain the surrounding edema and congestion. The incision was therefore enlarged and the abdomen explored. A mass the size of a kidney and resembling it in color could be felt and seen just above the incision. This was easily separated from several edematous epiploic appendixes, when it was seen to be a strangulated hemorrhagic omentum. The omentum was narrowed near its middle portion by a narrow pedicle which was twisted one and one-half turns (fig. 2). It was resected above the pedicle, and the raw end was closed with transfixion sutures. The abdominal viscera were otherwise normal on exploration.

Pathologic Examination.—Gross: The strangulated mass of omentum measured 14 cm. in length. It varied in width up to 5 cm. and in thickness up to 3 cm. The pedicle was 4 mm. in diameter. The one and one-half complete twists tended to return after untwisting. Distal to this point the omentum was

a dark, purplish red. It was firm and roughly oval, and most of the lobular markings were obliterated. The surface was covered with a thin layer of fibrin.

Microscopic Examination.—Blood was infiltrated throughout the fat lobules. Some of the blood vessels were thrombosed. At the upper end of the pedicle, there was considerable fibrous tissue with numerous round cells.

Postoperative Course.—The patient made an uneventful convalescence and was discharged from hospital on the twelfth day. One year after operation, he was free from any complaint and was doing his regular work.

In group 2, torsion has been reported in three patients (34, 43 and 44) who had old adhesions so that there were two fixed points of the omentum. The rarity of torsion with adhesions alone speaks against this as a common predisposing factor.

Group 3 includes at least 75 to 80 per cent of all cases of torsion. In this group the torsion either occurred directly in the hernial sac or was intimately associated with it. In some instances, the omentum was bilocular.

In group 4, a few reported cases of torsion occurred with an existing or with a history of a preexisting hernia, but there was no evident connection. The hernia, however, may have been a predisposing factor.

In group 5, torsion occurred with tumors or cysts present in the omentum. Prutz [1] found that torsion occurred with sarcomas, secondary carcinomas and echinococcus cysts present.

In group 6, abdominal tumors, abnormal changes in the viscera, pregnancy or other complications were present and might have influenced the development of a torsion. Bubis [2] has reported torsion associated with pregnancy; Frederick [3] found torsion of the omentum adherent to an ovarian cyst, while in a case of Payr's,[4] both the cyst and the omentum were twisted.

The first report of torsion of the omentum is credited by Mauclaire [5] and Aimes [6] to Marchette in 1851; Oberst [7] reported another case in 1882. Both cases, however, were associated with hernia.

The first case of torsion unassociated with hernia was reported in 1899 by Eitel.[8] Only the twenty-six cases in group 1 are analyzed here.

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1. Prutz, W.: Die Netztorsion, Deutsche Chir. **46**, K 1913.
2. Bubis, J. L.: Torsion of the Great Omentum During Pregnancy, Surg. Gynec. Obst. **28**:33 (Jan.) 1919.
3. Frederick, C. C.: Am. J. Obst. **56**:742, 1907.
4. Payr, E.: Ueber die Ursachen der Stelldrehung intraperitoneal gelegenir Organe, Arch. f. klin. Chir. **68**:501, 1902.
5. Mauclaire: Torsion de l'epiploon, Bull. méd. **24**:1195, 1910.
6. Aimes: La Physiologie du grand epiploon, Progrès méd. **34**:425, 1919.
7. Oberst, quoted by Pretzsch.
8. Eitel, G. G.: A Rare Omental Tumor, M. Rec. **55**:715 (May 20) 1899.

ETIOLOGY OF PURE TORSION OF THE OMENTUM WITHOUT HERNIA, ADHESIONS OR NEW GROWTHS

Sex.—Eigteen cases of torsion were stated to be found in males; six in females.

Age.—The average age at which torsion occurred was 38 years; the youngest patient was 1 year of age, and the oldest, 63. Two cases occurred in the first two decades, seven in the third, four in the fourth, six in the fifth and sixth, and only one in the seventh.

Factors Producing Torsion.—The Narrowing or Formation of a Pedicle on the Omentum. Payr believed that a narrow pedicle or bulbous tip predisposed to torsion, which resulted usually from hyperemia of the veins with rotation around the tense arteries. He produced spontaneous torsion of the omentum experimentally by the formation of gas cysts from 0.4 to 0.5 cm. in diameter, and also by the insertion of small pieces of cork and paraffin. Torsion was produced, he thought, by the shorter arteries acting as a mesentery which, following a hyperemia of the veins, produced a spiral out of the convex side. He demonstrated how torsion of a rubber tube occurred under similar conditions. He said that factors favoring torsion were similar in a distended appendix and might occur in any pedunculated organ.

Cunningham [9] described the omentum as usually lying more in the left hypochondrium with the left border extending over the descending colon and undergoing less motion than the right side, which frequently presents one or more processes. Anatomic variations in the blood vessels may predispose to a pedicle. Cowell [10] found that the blood supply of the omentum arose from the long epiploic branches of the right and the left gastro-epiploic arteries. These course over the transverse colon, not between the anterior layers of the omentum, but in little peritoneal folds on mesenteries of their own. They form an arch at the lower border of the omentum. Posteriorly, a few branches from the middle colic artery reach the lower portion of the omentum.

The congenital formation of a pedicle should be considered in group 1, since adhesions are not present. Broca [11] has reported torsion in a young infant, and it also occurred once in infancy in group 1. Numerous anomalies of the omentum have been described. Draper and Johnson [12] emphasized the importance of congenital omental bands which, by roentgenologic examination, they demonstrated produced

9. Cunningham: Human Anatomy, quoted by Cowell.

10. Cowell, E.: Abdominal Torsion of the Omentum, Brit. J. Surg. **12**:738 (April) 1925.

11. Broca, quoted by Thevenard.

12. Draper and Johnson: The Pathologic Omentum, J. A. M. A. **88**:376 (Feb.) 1927.

angulation and pressure on portions of the colon. They found them on both sides in the majority of instances and in the absence of previous inflammatory disease. Bierman and Jones [13] reported a congenital accessory omentum arising from the lesser curvature of the stomach; it hung over and produced a filling defect of the stomach in one case.

In two of the cases reviewed the strangulated portion was believed to be an accessory omentum; in both of these, the pedicle was narrow. One occurred in a thin woman, while the other occurred in an obese patient. Recently, I operated on a man, aged 58, who had acute obstruction of a loop of jejunum due to omental bands. These were formed apparently by a congenital division of the end of the omentum into two portions, one of which passed through an anomalous opening in the mesentery and fused solidly with the other beneath the superior mesenteric artery. There was no obesity of the omentum. Following resection of the omentum, the uncomfortable fulness on eating that had been present all his life and the recent symptoms of obstruction immediately cleared up.

Obesity may be a predisposing factor to formation of pedicles through the tendency to a bulbous enlargement of the omentum with a narrowing at the base following unequal infiltration with fat. The production of a pedicle might easily result from pressure at the neck of the sac in a prolonged herniation of the omentum. Other factors predisposing to a pedicle in the omentum might be: constriction about the waist as from a belt, constant abdominal pressure in some special work, a prolonged sitting position in obese persons and previous inflammation leaving a scar in the omentum, which might also cause unequal deposits of fat.

Hyperemia: Hyperemia of the veins with stasis and engorgement distally may be considered the chief activating factor in producing torsion, but this action probably occurs only in the presence of a pedicle. Numerous factors might produce a hyperemia of the omentum, such as cardiac weakness, traction by adhesions, pressure from within or without the abdomen, heavy or unusual work, coughing, sudden muscular contraction of the abdominal muscles, unusual positions of the body and tumor or obesity of the omentum. Hyperemia might also result from inflammation in a contiguous area, such as that caused by appendicitis [14] or a preceding abortion.[15]

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13. Bierman and Jones: A Third Omentum, Surg. Gynec. & Obst. **36**:708 (May) 1923.

14. Erdmann, J.: Ann. Surg. **88**:252, 1921.

15. Riedel: Ueber gedrehte Netzgeschwulste mit und ohne vorgangren Bruch, München Med. Wchnschr. **52**:2257 (Nov. 21) 1905 (two case reports).

While torsion may also develop from a number of causes, it is evident that a narrow pedicle may exist for a long time before rotation occurs.

Obesity: Fat infiltration may predispose to torsion by development of a pedicle with a bulbous tip. It may also produce changes in the blood vessels leading to stasis or vascular tension. In the eight cases in group 1 in which general obesity of the patient was noted, there was little narrowing at the site of the torsion in seven, but in one the pedicle was small. In ten other cases, the strangulated portion of the omentum was described as a fat round mass.

Obesity of the omentum probable predisposes to torsion, since obesity was more marked in the cases in which there was no definite pedicle.

Inflammation of the Omentum or Neighboring Viscera: Inflammation of the omentum or neighboring viscera may lead not only to pedicle formation, but may produce hyperemia. Acute or chronic inflammation may result in scars, constricting bands, adhesions with a second fixed point to the omentum or to incarceration in a hernial sac. Schoenholzer [16] stated that torsion of the omentum did not occur without previous inflammation. Skeel [17] reported chronic appendicitis as being present in eight of ten reviewed cases, and he thinks that this predisposed to torsion. Inflammation was probably not an important factor in group 1.

Trauma or Pressure: An acute trauma of the omentum from either within or without the abdomen may produce torsion. In Eitel's patient, torsion resulted from chronic pressure of a box. My first patient fell on the upper part of his abdomen. Bazy [18] has suggested the pressure of intestinal peristalsis as exciting torsion. Pressure of an abdominal tumor might produce it, such as an ovarian cyst, or a pregnancy, as in Bubis' [2] case. Sudden contraction of the abdominal muscles as well as a blow applied with the muscles in relaxation have been noted. In Kraske's [19] case, torsion followed an operation in the Trendelenburg position. Taxis on a strangulated hernia has been noted by Lejars [20] and others.

Heavy Work or Unusual Physical Exertion: Torsion followed heavy or unusual work in Mullen's [21] and in Eitel's [8] cases, but the symptoms were slow in developing; usually the onset was of short dura-

16. Schoenholzer, quoted by Thevenard.

17. Skeel, R. E.: Intra-Abdominal Torsion of the Omentum Without Hernia, Am. J. Obst. **56**:792, 1907.

18. Bazy, quoted by Thevenard.

19. Kraske, quoted by Aimes.

20. Lejars: Semaine méd. **27**:73, 1907.

21. Mullen, T. F.: Torsion of Great Omentum, Surg. Gynec. Obst. **40**:635 (May) 1925.

tion. It followed horseback riding in Ill's [22] case; in Pretzsch's [23] case, it followed dancing, and in Cullen's [24] case, the patient had been violently whirled around. In Kohler's [25] patient, torsion followed the lifting of a heavy object. Thevenard [26] reported an instance following bicycle riding. Wiener [27] believed that attacks of coughing were a cause in one case. In my second case, the onset followed unusual physical exertion.

Another factor in torsion, which obtains in group two is the formation of a second fixed point. This may be due to the fixation of the omentum by adhesions or to incarceration in a hernial sac. The mechanism of two fixed points may be compared to a folded handkerchief with two corners held taut, allowing the third corner to rotate.

PATHOLOGY

The pedicle was at the base of the omentum with complete strangulation in eleven cases. There was a narrow pedicle in two of these, slightly narrowed to the size of the thumb in four, doubtful narrowing in three, no pedicle in one and twisting of the entire omentum in one. Obesity was noted in the four cases in which there was only a slight narrowing at the base.

The pedicle in the great omentum was not at the base, and the torsion strangulated only a part of it in thirteen cases. There was a pedicle the size of a pencil or smaller in nine of these, a slight narrowing in two, a doubtful narrowing in one and no pedicle in one which contained a twist over the entire distal half of the omentum. Three of these patients were obese, including the two instances of only slight narrowing. In the only patient noted as thin, there was a narrow pedicle with eight twists.

The pedicle was narrow in 50 per cent of the twenty-six cases, and varied in size from that of a knitting needle to that of a lead pencil. There was some narrowing in 23 per cent, doubtful narrowing in 15 per cent and no narrowing in 12 per cent. There was an average of three complete twists of the pedicle in sixteen cases. In one of Riedel's [15] cases there were two separate points of constriction of the omentum with torsion of each.

The pedicle was usually short, but in a few instances it was several centimeters long. The twist extended over the entire omentum in two

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22. Ill, E. J.: Am. J. Obst. **56**:742, 1907 (two case reports).
23. Pretzsch, E.: Ueber die Torsion des Netzes, Beitr. z. klin. Chir. **48**:118, 1906.
24. Cullen: Torsion of an Accessory Omentum Producing Symptoms Simulating Appendicitis; Recovery, Bull. Johns Hopkins Hosp. **16**:401, 1905.
25. Kohler, R.: Netztorsion, Arch. f. klin. Chir. **3**:514, 1918.
26. Thevenard: Torsion aigue de l'epiploon, Paris chirurg. **3**:38, 1911.
27. Wiener: Ann. Surg. **32**:648, 1900.

cases. The entire omentum was strangulated in 42 per cent, a portion of the omentum in 50 per cent and an accessory omentum in 8 per cent. Where only a part was involved, it was noted that it was the distal half in five cases, the right free border in one, the right lower corner in one and the right half in the case that was associated with acute appendicitis. The pedicle arose by a slender talk of blood vessels on the free edge of the omentum in one and in an undesignated part in four instances. In the two cases of accessory omentum, this arose from the transverse colon at the right of the main omentum.

The size of the strangulated mass of omentum varied. It was described as fat in 69 per cent of the cases. In eleven instances in which measurements were reported, it averaged 12 by 8 by 3 cm. in size. It usually appeared dark red or purple and was frequently in a state of red infarction. The veins were only occasionally reported distended. The consistency was firm, almost as if it were frozen.

Three stages of changes have been described in the omentum. The first stage is congestion and venous stasis; the second, the most frequently noted, is extravasation of blood, infarct formation and thrombosis of blood vessel as in Kohler's [25] case, and the third stage is necrosis. Only in Simon's [28] and Lefebvre's [29] cases were gangrenous areas reported. Areas of increased connective tissue formation with exudation of white blood cells and stasis were noted in Wiener's [27] and in my cases.

The appendix was found acutely inflamed in Scudder's [30] case, slightly congested in two, thickened in one, containing a fecalith in one, obliterated in one and normal in eight other cases. Congestion in the appendix could be excited by the torsion or vice versa, although there were no adhesions of the omentum to the appendix in group 1.

Edema and thickening of the peritoneum were present over a large area near the strangulated omentum in my two cases, together with a marked hyperemia which also involved the abdominal muscles. In a number of instances described, there was an agglutination with recent fibrinous adhesions of the omentum to the gallbladder, the liver or the diaphragm or to portions of the intestine and the ascending colon. The epiploic appendixes were edematous and loosely adherent to the omentum by fibrin in my two cases. Baldwin [31] described a local peritonitis.

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28. Simon, O.: Intra-abdominal Netztorsionen, München Med. Wchnschr. **52**: 1979 (Oct.) 1905.

29. Lefebvre, C.: Torsion du grand epiploon, Bull. et mém. Soc. de chir. de Paris **49**:175, 1923.

30. Scudder, C. L.: Intra-Abdominal Torsion of the Entire Great Omentum, Ann. Surg. **40**:916 (Dec.) 1904.

31. Baldwin, J. F.: A Contribution to the Study of Intra-Abdominal Omental Torsion, Ann. Surg. **36**:940 (Dec.) 1902.

Free fluid was noted in the peritoneal cavity in eight cases; it was bloody in five. The amount varied from a few ounces to 1,500 cc. in Simon's case.

SYMPTOMS

There were preexisting abdominal symptoms in only two cases of this group, although in a number of cases of torsion associated with hernia, previous symptoms have frequently been noted. In Hinton's [32] case of torsion, there was a history of indigestion for several years, and at operation for torsion, gallstones were discovered. In Skeel's [17] case, there had been symptoms of the stomach for one year, probably due to drinking to excess. Apparently the previous symptoms were in no instance referable to the omentum, although in the majority of this group, there was a narrow pedicle which had evidently existed for some time. Previous symptoms of chronic appendicitis were completely lacking.

The duration of symptoms of torsion from the onset to the operation varied from one to seventeen days, the average duration being five days in nineteen cases, excluding Eitel's [8] case of over four weeks' duration. The course of symptoms from the onset was usually noted as gradual and progressive over a period of several days. The first complaint usually was a discomfort in the abdomen, with development of severe and cramplike pains later. In Riedel's [15] case, there was vomiting eleven days before pain developed. In three other cases (Simon [28] Nixon,[33] Cowell [10]), pain was not severe until after from four to ten days. The onset was noted as sudden, with acute development of symptoms in six cases.

Vomiting was noted as present in ten cases and absent in ten. It occasionally occurred before pain developed. In two cases (Scudder,[29] Noble [34]), it stopped after the onset. In two others (Simon,[28] Mullen,[21] it occurred late, together with the increasing symptoms. In three cases (Stewart,[35] Skeel,[17] Riedel's [15] second case), vomiting occurred throughout the entire period. Pain and tenderness were present in all except Eitel's case. The location of pain was noted as being in the right lower quadrant in twelve cases, although in one (Cowell) it began in the epigastrium. It was located in the right side in seven cases and in the right upper quadrant in two cases.

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32. Hinton, J. W.: Torsion of Great Omentum Producing Symptoms Simulating Acute Appendicitis, Arch. Surg. **13**:507 (Oct.) 1926.

33. Nixon, J. W.: Torsion of the Great Omentum with Report of a Case, Texas State J. Med. **20**:659 (April) 1925.

34. Noble, T. B.: Intra-Abdominal Torsion of the Omentum, Am. J. Obst. **49**:364, 1904.

35. Stewart: Volvulus of the Omentum, J. A. M. A. **42**:767, (March 19) 1904.

On physical examination, a palpable mass was noted in six cases and was absent in twelve. It was palpated in three cases in the right lower quadrant, once in the hypogastrium and once in the right upper quadrant. While the pain was to the right of the umbilicus in Riedel's first case, there was a mass in the left lower quadrant palpable by vaginal examination. In two instances, there was a subjective sensation of a mass.

Rigidity and muscle spasm were noted in twelve cases; the abdomen was boardlike in four cases. Rigidity was absent in Cullen's [24] and in Grove's [36] cases. Tenderness was noted in fourteen cases and was exquisite in eleven of them.

In one case (Lefebvre) with an acute onset, hyperesthesia was not noted. Diarrhea was noted in one case (Skeel); constipation was noted occasionally. The pulse rate varied from 72 to 130, averaging 96 in ten cases. The temperature varied from a subnormal to 101.5 F. and averaged 99.6. The white blood count varied from 9,000 to 17,600 and averaged 14,000 in seven cases. There were 74 per cent of polymorphonuclears in one case.

DIAGNOSIS

The diagnosis of torsion of the omentum without hernia is difficult. It should be considered in an obese middle-aged man, with acute tenderness and an increasing cramplike pain in the right side of the abdomen, continuing for several days following some unusual or violent physical exertion, especially in the absence of nausea or vomiting. The exquisite tenderness which is out of proportion to the other symptoms, and cramplike pains increased by movement, are the most characteristic symptoms. Rigidity and muscle spasm, although usually not marked, may prevent the palpation of a mass. The temperature and pulse rate are not high, and the white blood count is lower than might be expected from the symptoms. The presence of bloody abdominal fluid should make one think of torsion.

The preoperative diagnosis was acute appendicitis in eighteen cases, acute cholecystitis in three, internal hernia of the omentum in one and a hydatid cyst in one. The appendix in one instance was thought to be located high up and in another to be associated with an abscess.

PROGNOSIS

In group 1, death occurred once from pneumonia, six days after operation (Lefebvre) in a man, aged 55, with a pulse of 130 on entrance and complete necrosis of the omentum. A postoperative acute dilatation of the stomach and pneumonia occurred in one of my cases. In review-

36. Groves, W. R.: Torsion of an Omental Tag, M. J. Australia **2**:697 (Nov. 21) 1925.

ing forty-four cases of all groups not accompanied by hernia, including tumors, Pretzsch reported six deaths, or a mortality of 14 per cent.

In torsion of omentum, including cases in which hernia was present, Corner and Pinches [37] reported eight deaths in fifty-three cases, or a mortality of 13 per cent. Pretzsch reported a fatal case of general purulent peritonitis of Moresco's, and he thought that the condition was probably embolic in origin. He also reported a case of Eiselsberg's in which postoperative bleeding from the stomach followed resection of the omentum. Deaths have been reported from pneumonia, infection of the gangrenous mass of omentum, obstruction of the intestine and pulmonary embolism.

The appendix has been found acutely inflamed. Mechanical obstruction of the transverse colon by torsion of the omentum with passage of blood and mucus was reported by Bookman [38] in a case in which hernia was present.

Later unfavorable symptoms following removal of the entire omentum have not been noticed, according to Pretzsch.

TREATMENT

Early operation is indicated and should usually permit the exploration of the entire abdomen. There is danger of overlooking a strangulated omentum due to the edema and hyperemia of the neighboring viscera. When congestion of the appendix or other viscus is not sufficient to explain the physical and laboratory observations, a further exploration of the abdomen should be made.

The omentum should be resected, even if it is possible to untwist it. It was resected in all but one case. The appendix was removed in twelve cases and examined and left as normal in two. It is possible to overlook a second torsion of the omentum higher up. The blood vessels should be ligated by transfixion, as there is danger of hemorrhage, and the edges should be carefully sutured. When the pedicle is found at the base of the omentum, one should carefully avoid interference with the blood supply to the colon. The omentum should be examined during every abdominal or hernial operation if possible, especially in obese persons. When there is a tendency to formation of a pedicle, it should be resected and any omental bands due to anomalies or adhesions should be cut as a prophylaxis against torsion or intestinal obstruction. Following any abdominal operation, one should carefully spread out the omentum.

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37. Corner and Pinches: Med. and Clin. Tr., London **88**:611, 1905.

38. Bookman: Complete Torsion of the Great Omentum, Am. J. Surg. **29**:304, 1915.

SUMMARY

1. Torsion of the omentum may occur in the absence of hernia or any pathologic condition and without previous abdominal symptoms.

2. There is almost always evidence of a preexisting pedicle, which may be congenital in origin.

3. Obesity of the omentum is present in the majority of cases, and it is probably a frequent predisposing factor both in the formation of a pedicle and in the torsion.

4. Hyperemia may be the usual exciting factor in torsion, although trauma or unusual physical exertion may initiate it.

5. Prophylactic resection of a pedunculated omentum and freeing of adhesions is usually advisable.

6. Early operative resection of the strangulated omentum should be performed.

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40. St. Thomas Hospital Reports **34**:283-284, 1905. Two case reports. (One case probably the same one reported by Corner and Pinches.)

41. Syme, quoted by Cowell: Intercolon, M. J. Australasia, 1902, p. 444.

42. Brown, F. R.: A Case of Abdominal Torsion of the Omentum, Brit. M. J. **1**:183 (Jan. 30) 1926.

43. Fuller, W.: Intra-Abdominal Rotation of the Great Omentum, Unaccompanied with Hernia, Surg. Gynec. Obst. **7**:231 (Aug.) 1908.

44. D'Allaines and Rouffiac: Torsion intra-abdominale du grand epiploon sans hernia, Bull. et mém. Soc. Anat. de Paris **20**:327, 1923.

ADENOMYOMA OF THE STOMACH *

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Few reports of cases of adenomyomas of the stomach are found in the literature. Many of the references to this condition are included in discussions of other closely related heterotopias of the digestive tract, such as accessory islands of pancreatic tissue. Although most of these heterotopias are only of academic interest, adenomyomas have a definite clinical importance because of their closer approach to a neoplastic type of growth, the actual presence of a tumor mass, the similarity in appearance to carcinoma at operation and the possibility of clinical symptoms.

REVIEW OF THE LITERATURE

In 1903, Magnus-Alsleben [1] reported a series of five adenomyomas of the stomach found accidently at autopsy. All were quite small and situated near the pyloric ring. Microscopically, all showed alveoli of various types surrounded by smooth muscle bundles. Some of the alveoli were relatively undifferentiated, others resembled Brunner's glands, and still others were similar to pancreatic alveoli. In all, the glands of the tumor connected at some point with Brunner's glands of the submucosa.

In 1909, Gruzdeff [2] reported the first adenomyoma of the stomach to be observed clinically. The patient, a woman, aged 36, had been feeling ill for about a year. During the six weeks previous to operation, she had been treated for severe anemia and "dyspepsia." Gastric analysis demonstrated free hydrochloric acid. A tumor mass about the size of an apple was present in the right upper quadrant of the abdomen. It was painless, freely movable and smooth. The preoperative diagnosis, based on the progressing cachexia and presence of the tumor, was cancer of the stomach. At operation the tumor was found embedded in the posterior wall of the stomach near the pylorus. It was removed with a narrow margin of the wall of the stomach. The patient made a complete recovery and was without symptoms six

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1. Magnus-Alsleben, E.: Adenomyome des Pylorus, Virchows Arch. f. path. Anat. **173**:137, 1903.

2. Gruzdeff, D. A.: A Rare Case of Adenomyoma of the Stomach, with Heterotopia of the Epithelium of the Mucous Membrane, Med. Obozr. **71**:805, 1909.

months after the operation. Pathologic examination showed the tumor to consist of alveoli surrounded by smooth muscle bundles. The epithelial lining of the alveoli resembled that of the stomach mucosa and was single-layered. The alveoli did not open into ducts but formed closed cystic cavities. A connection between the alveoli of the tumor and the epithelium of the stomach was not demonstrable. Evidence of malignancy was not found.

In 1925, Stewart and Taylor [3] described four cases. One was found at autopsy, the other three at exploratory operations. In the three latter cases, the adenomyoma constituted the only abnormal observation. In one case in which gastric tabetic crises was later suspected, the patient was unrelieved. The two remaining patients complained of epigastric pain which came on several hours after meals and which was relieved by food. A roentgen-ray examination was performed on one of the patients, but a pathologic condition was not revealed. Both patients were relieved by operation. All of the tumors were small and located near the pyloric ring. Microscopically, they showed alveoli of the Brunner's gland type, alveoli of the pancreatic type and undifferentiated alveoli. All were surrounded by smooth muscle strands. Duct-like structures were also present.

REPORT OF A CASE

History.—Miss E. J., a practical nurse, aged 36, entered the medical service of the University of California Hospital on March 1, 1926, with a complaint of vomiting and epigastric pain. Her family and past history were essentially negative. About five years before (January, 1921), she had had attacks of vomiting without nausea, followed by gnawing, nonradiating epigastric pain not related to meals and unrelieved by food or sodium bicarbonate. The vomited material had frequently contained fragments of food eaten one or two days previously, but never blood or coffee-ground material. Other gastro-intestinal symptoms, except belching, had been absent. The attacks had occurred several times a day at intervals of from a few days to several weeks. She had consulted a physician, who, after taking roentgenograms, made a diagnosis of "peptic ulcer." Dietary regimen had not given relief, a small duodenal ulcer about 1 inch from the pylorus had been found and at operation (October, 1922) a posterior gastro-jejunostomy had been established. After an uneventful convalescence, she had remained symptomless for about a year. In January, 1924, her gastro-intestinal symptoms had recurred while she was convalescing from a postinfluenzal mastoiditis. Two months before entering the University of California Hospital, she consulted one of us. Dietary regimen and alkalis failed to relieve her symptoms. During this period, the pain was almost constant and frequently kept her awake at night.

Examination.—During examination the patient was found to be of a neurotic type, given to exaggeration of all her symptoms, easily upset mentally and with a tendency toward moroseness. The physical examination, except for marked diffuse tenderness over the entire epigastrium, was otherwise negative. A blood

3. Stewart, M. J., and Taylor, A. L.: Adenomyoma of the Stomach, J. Path. & Bact. **28**:195, 1925.

count showed 95 per cent hemoglobin and 4,600,000 red blood cells. The results of urinalysis were negative. Occult blood was not present in the stool. Gastric analysis after an Ewald meal showed free hydrochloric acid to 35 in one hour and total acid to 80 in two hours. The blood Wassermann reaction was negative. Roentgenologic examination showed a small high stomach emptying through a wide gastro-enterostomy opening. There was considerable spasm of the pylorus and first portion of the duodenum. Defects were not noted. The conclusion possibly ulceration of the jejunum or at the pylorus was present.

Fig. 1.—Entire tumor showing diffuse scattering of alveoli and ductlike structures throughout the submucosa and muscularis of the stomach. Reduced from a magnification of × 20.

Operation.—The patient was transferred to the surgical service on March 5, 1926, for an exploratory laparotomy. The preoperative diagnosis was jejunal ulcer with pylorospasm. She was operated on by one of us on the following day. Nitrous oxide and ether anesthesia was used. The incision was made over the left rectus muscle. Adhesions of the omentum to the anterior abdominal wall at the site of the former scar were present. The gallbladder was slightly thickened. The foramen of Winslow was open. The stomach was approximately normal in size, and the previous posterior gastrojejunostomy was in excellent condition, except for herniation of a knuckle of jejunum through the opening in the trans-

verse mesocolon which allowed some distortion of the lumen of the bowel. A small scarlike area was found on the anterior surface of the pylorus. Gastrostomy revealed thickening at this point with an intact mucous membrane. The thickening was believed to represent the scar of a healed ulcer. A pylorectomy was performed, and the stomach was closed just distal to the previous gastrojejunostomy. The knuckle of jejunum which protruded through the rent in the transverse mesocolon made at the previous operation was withdrawn, and the rent was repaired by reattaching the stomach to the transverse mesocolon. The patient was returned to the ward in good condition. Recovery was uneventful.

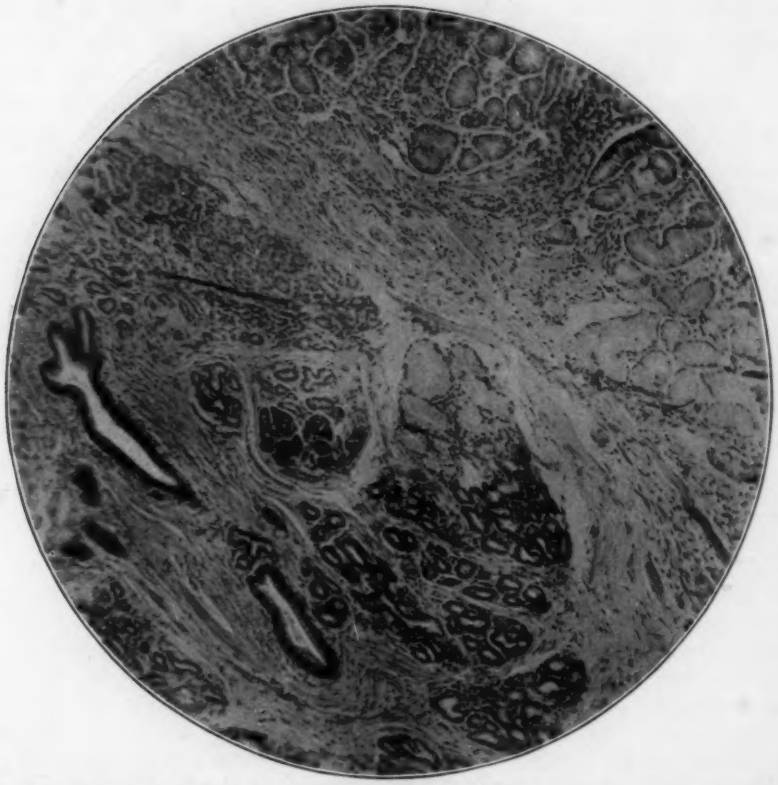

Fig. 2.—Portion of tumor immediately beneath the mucosa of the stomach. Ductlike structures, undifferentiated alveoli, and alveoli of the Brunner gland and pancreatic type are seen. Reduced from a magnification of × 80.

Pathologic Report.—The specimen consisted of the pyloric portion of the stomach. It measured 8 by 5 by 4 cm. and included about 2 cm. of the duodenum. The specimen had been turned inside out. The mucosa appeared essentially normal. About 2 cm. from the pyloric ring on the anterior gastric wall was a nodule 0.5 cm. in diameter. This nodule was covered by intact normal-appearing mucosa. Sections through this area showed a thickening of the wall of the stomach involving chiefly the muscularis. Grossly, the appearance of the cut surface suggested an old callous ulcer.

Microscopic examination of sections through the nodule showed an intact, essentially normal, mucosa. The submucosa was thickened and fibrous. Numerous glands and ductlike structures were present in scattered groups throughout the submucosa and muscularis. Some of these glands resembled pancreatic acini, while others approached the type of the Brunner gland. Undifferentiated alveoli and intermediate types were numerous and made up the bulk of the tumor. These glandular structures appeared essentially normal in all respects except location. There was no suggestion of malignancy. Narrow strands of smooth muscle, apparently distinct from the muscularis of the stomach, surrounded these alveoli.

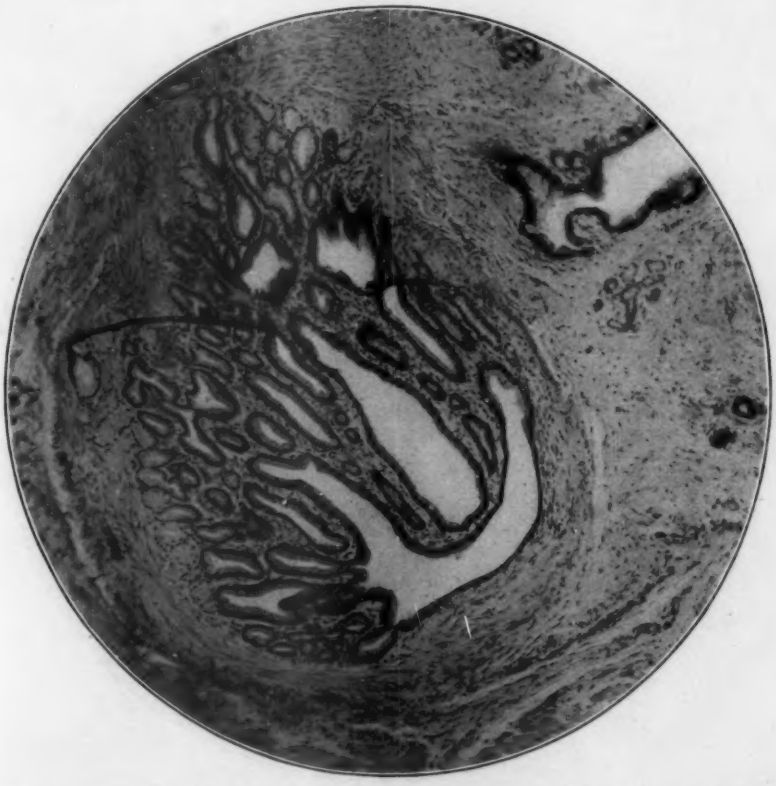

Fig. 3.—Ductlike structure surrounded by distinct bundles of smooth muscle. Reduced from a magnification of × 80.

The muscularis of the stomach showed some diffuse fibrosis and was moderately infiltrated with lymphocytes and plasma cells. The diagnosis was adenomyoma of the stomach.

Subsequent Course.—The patient left the hospital on March 22, 1926, and, against our advice, immediately returned to work. Lack of strength and recurrent attacks of vomiting finally compelled her to stop work. Roentgenologic examination showed gastric emptying by the gastrojejunostomy stoma as complete but slightly more rapid than normal. Four weeks of rest in the country allowed her to regain 12 pounds (5.4 Kg.) in weight and a corresponding amount of strength; it also gave her a happy outlook on life. Since that time she has had gastric

upsets at rare intervals, always preceded by dietary indiscretions; these attacks are characterized by cramplike pain in the upper part of the abdomen and vomiting for from four to twelve hours. She is at present otherwise well.

COMMENT

In all of the reported cases of adenomyoma of the stomach, the tumor has been located in the pyloric portion, usually within 3 cm. of the pyloric ring. At operation a definite localized thickening of the

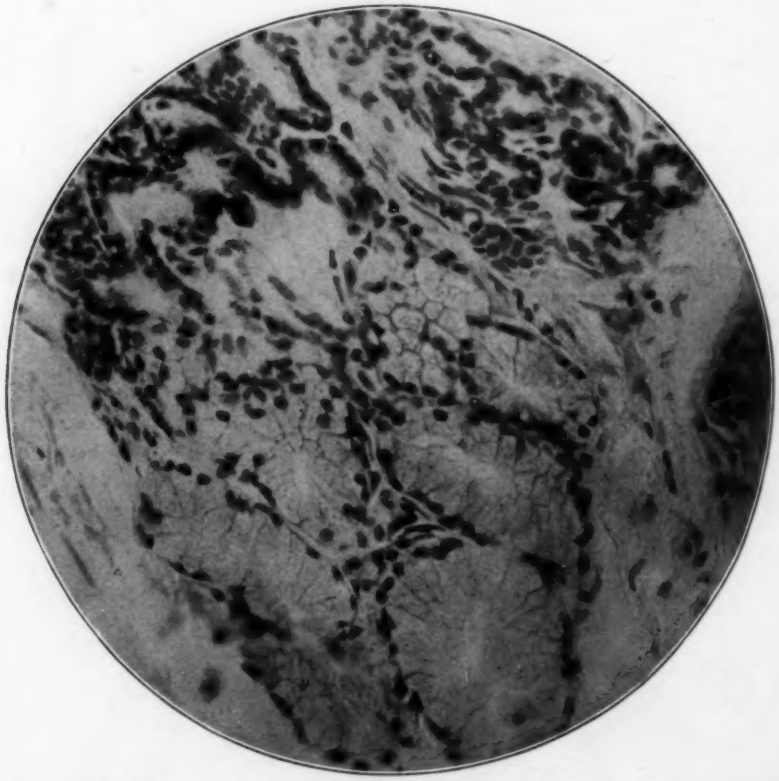

Fig. 4.—Brunner gland type of alveoli. Reduced from a magnification of × 320.

wall of the stomach is always palpable. This thickening may also be visible as an ill defined and somewhat irregular bulging of the peritoneal surface of the wall of the stomach. It thus closely resembles an early carcinoma of the pylorus from its peritoneal aspect. When the stomach is opened, the mucous membrane is seen to be intact over the tumor. This intact mucous membrane at once differentiates it from a carcinoma. A cut through the tumor shows the thickening to be localized in the submucosa and muscularis. Whorls of muscle fibers may be seen

entering the tumor mass. Pin-point cystic cavities are frequently visible; occasionally small plugs of secretion and epithelial debris may be expressed by pressure. When these cystic cavities are absent, the tumor may closely resemble the scar of an old callous ulcer.

Microscopically, glandular structures are found throughout either the submucosa and the muscularis or throughout both. These glandular structures are frequently grouped into nodules of varying sizes. Many alveoli appear relatively undifferentiated. These are lined by dark-

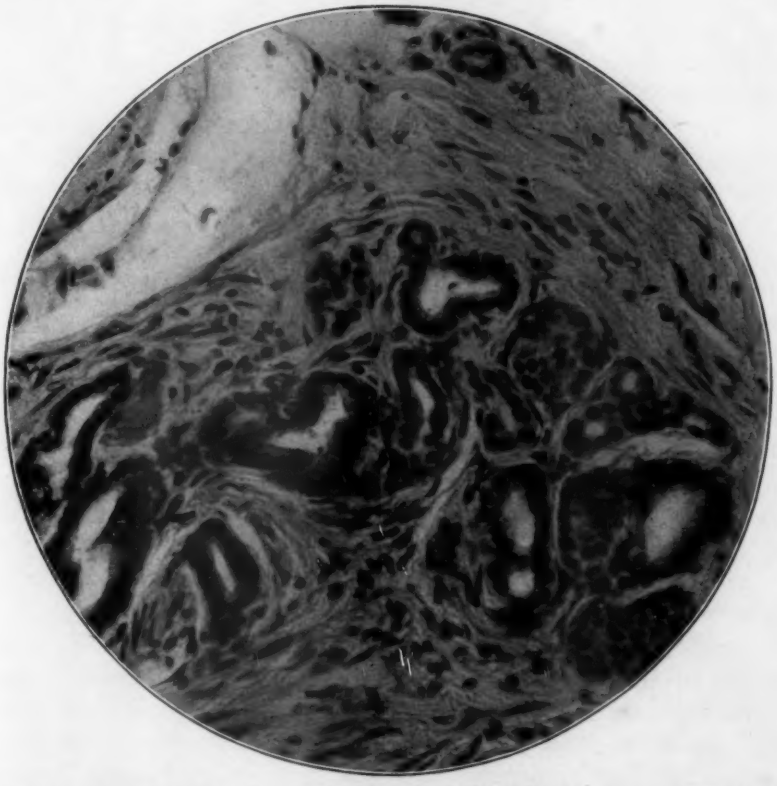

Fig. 5.—Undifferentiated alveoli. Reduced from a magnification of × 320.

staining cells with round to oval nuclei which ranged from cuboidal to columnar in shape. The cells, although undifferentiated and embryonic in character, appear active. They resemble the lining cells of the biliary and pancreatic ducts rather than gastric or intestinal mucosa. Differentiated alveoli resembling either pancreatic acini or Brunner's glands are also present. Usually all three types are found, but any one may dominate the picture. The alveoli open into ductlike structures which eventually communicate with the lumen of the stomach.

Occasionally ducts are absent, and the alveoli form closed cystic cavities of varying sizes. The cells lining these cystic spaces are undifferentiated and embryonic. Bundles of smooth muscle fibers surround the groups of alveoli. These muscle bundles follow the tumor alveoli and ducts into the submucosa and apparently constitute an integral part of the tumor. Intermingling of the muscle bundles of the tumor and the stomach muscularis occurs. Evidences of neoplastic tendency are most

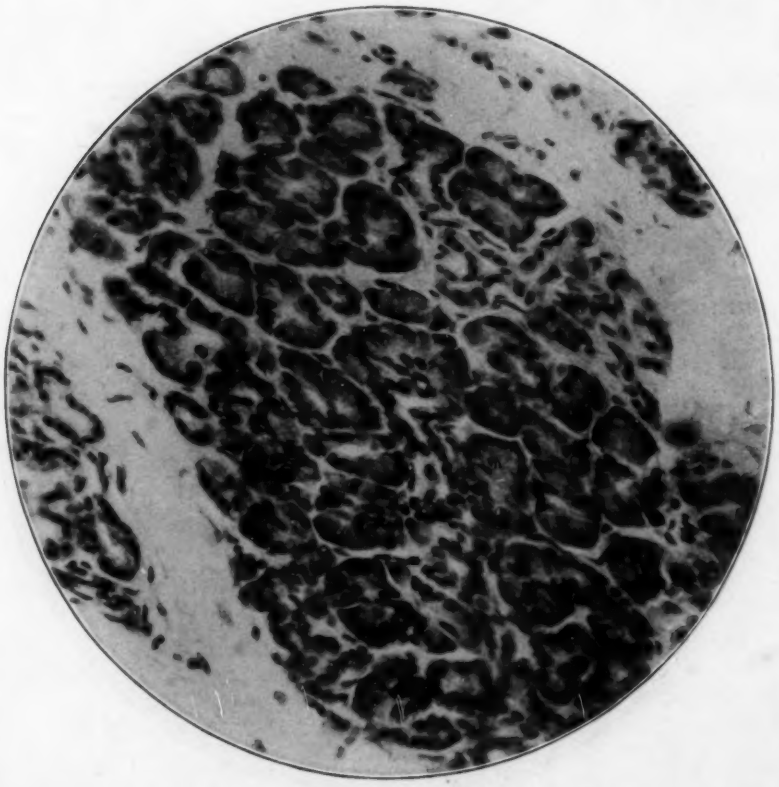

Fig. 6.—Nodule of pancreatic type tissue. Reduced from a magnification of × 320.

marked in the undifferentiated alveoli which may form relatively large nodular groups. These alveoli, although active-appearing, are apparently nonfunctional. The lining cells are always in single layers and are arranged in an orderly manner. Evidences of malignancy are absent.

Malignant degeneration of these tumors is possible, but no such cases are known. As a rule, accessory organs do not show a greater tendency toward malignant change than normal organs. Accessory

pancreases are relatively common, yet Nicholson [4] could find only three cases of carcinoma arising in an accessory pancreas. Adenomyomas show more tendency toward a neoplastic type of growth than accessory pancreatic nodules, but their rarity reduces to a negligible factor the possibility of their being the origin of a carcinoma. It must also be remembered that such an origin would be extremely difficult to prove. It would be necessary to discover the carcinoma before involvement of the gastric mucosa had occurred, and a carcinoma of such size would be discovered only accidently at exploratory operation or at autopsy following death from other causes.

Adenomyoma of the stomach represents one of the many heterotopias of the digestive tract. It is of congenital origin and develops from epithelial buds or diverticuli. Accessory pancreases arising from similar epithelial buds form the commonest heterotopia of this type. Unlike the pancreatic heterotopias, adenomyoma tends to remain wholly or in part undifferentiated, or to differentiate in two directions, that is, toward both the Brunner gland and the pancreatic types. As a result, most adenomyomas show three types of glands: undifferentiated, Brunner gland type and pancreatic type. The undifferentiated alveoli usually predominate and approach a neoplastic type of growth. Strands of smooth muscle are associated with the tumor. In some cases the connection of the epithelial bud with the lumen of the stomach become lost, and either single or multiple cystic cavities result. The latter heterotopias, we believe, can be considered adenomyomas only if the cystic spaces are lined by epithelial cells similar to those of the undifferentiated type of alveoli and show the same proliferative tendency. The adenomyoma reported by Gruzdeff is an example of this type.

Lauche reserves the term adenomyoma for those heterotopias in which differentiation does not lead to the formation of pancreas-like acini. He classifies heterotopias similar to the condition in our case and in the cases reported by Magnus-Alsleben, Stewart and Taylor as incompletely differentiated accessory pancreases. Although intermediate forms between true accessory pancreases and adenomyomas occur, we believe with Stewart and Taylor that the presence of undifferentiated alveoli with neoplastic tendencies justifies the inclusion of these growths in a separate group as adenomyomas.

The clinical significance of these adenomyomas depends partly on the necessity for differential recognition at operation and partly on the possibility of clinical symptoms. The exact symptomatology is open to doubt. Most of the reported cases were discovered accidentally at autopsy. Those observed clinically have not, on the whole, been

4. Nicholson, G. W.: Studies in Tumor Formation, Guy's Hosp. Rep. **73**:37, 1923.

thoroughly studied. The case reported by Gruzdeff is remarkable because it is the only one in which the symptomatology simulated a malignancy. The severe anemia, progressive cachexia and palpable tumor all led to a clinical diagnosis of malignancy. Gastric symptoms described as "dyspepsia" were present. Complete relief followed operative removal. One of the cases reported by Stewart and Taylor gave a history of epigastric pain associated with vomiting. The pain occurred every two or three weeks in attacks lasting a week or ten days. Operation failed to relieve the symptoms, and the attacks were later diagnosed as tabetic crises. Their other two patients complained of pain coming on several hours after meals and relieved by food; vomiting did not occur. Results of a roentgen-ray examination performed in one of the cases were negative. Operation did not disclose any other pathologic gastric condition. Complete relief followed the excision of the adenomyomas. In our case the recurrence of the symptoms after operation suggested some cause other than the adenomyoma.

Judging from the reported cases, the symptomatology of adenomyoma is vague and indefinite. In rare cases, it may simulate that of an ulcer or even of a malignancy. In most cases, the symptoms are slight and probably depend chiefly on interference with the motor function of the stomach.

SUMMARY

1. Adenomyoma of the stomach represents one of the rarer heterotopias of the digestive tract.

2. The importance of gastric adenomyoma depends chiefly on the necessity for differential diagnosis at exploratory operations.

3. Definite symptomatology ordinarily is not present.

4. Treatment should consist of simple excision with a narrow margin of the wall of the stomach.

EXPERIMENTAL CHRONIC DUODENAL OBSTRUCTION

CHANGES IN THE BLOOD AND OTHER PATHOLOGIC CHANGES *

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Our primary object in the experimental production of chronic stenosis of the duodenum was to determine whether there was any relationship between intestinal stasis and alterations in the blood. Clinically, Löwenberg [1] and Olivet [2] found an increase in the bacterial content of the duodenum in cases of pernicious anemia. Experimentally, Seyderhelm [3] claimed to have produced a pernicious anemia-like picture in dogs with chronic obstructions of the small intestine. Brown and his co-workers [4] reported the occurrence of "toxic nephritis" in a series of cases of chronic duodenal obstruction. Functional studies of the kidneys indicated renal insufficiency. Chemical examination of the blood revealed an increase in the urea nitrogen and creatinine, a high carbon dioxide carrying capacity and a low chloride content.

In previous papers,[5] a new technic for producing chronic stenosis of the duodenum was described. The duodenum was chosen as the site of the obstruction because, normally, this segment of the intestine is relatively free from bacteria, and the rate of motility is more rapid than in any other part of the intestinal tract. As a result of the obstruc-

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* Preliminary report of part of this work appeared in the Proc. Soc. Exper. Biol. & Med. **24**:590, 1927.

1. Löwenberg, W.: Pathologische Bacterienansiedlung im Duodenum und ihre ursächlichen Faktoren, Klin. Wchnschr. **5**:548 (March 26) 1926.

2. Olivet, J.: Bakteriologie des Duodenums, Klin. Wchnschr. **5**:307 (Feb. 19) 1926.

3. Seyderhelm, R.: Die Bedeutung des Dünndarms für die Genese der perniziösen Anämie, Klin. Wchnschr. **3**:568 (April 1) 1924. Seyderhelm, R.; Lehman, W., and Wichels, P.: Experimentelle Intestinale perniziöse Anämie beim Hund, Klin. Wchnschr. **3**:1439 (Aug. 5) 1924.

4. Brown, G. E.; Eusterman, G. B.; Hartman, H. R., and Rowntree, L. G.: Toxic Nephritis in Pyloric and Duodenal Obstruction, Arch. Int. Med. **32**:425 (Sept.) 1923.

5. Berg, B. N.; Meleney, F. L., and Jobling, J. W.: Experimental Chronic Duodenal Obstruction, Arch. Surg. **14**:752 (March) 1927. Meleney, F. L.; Berg, B. N., and Jobling, J. W.: Experimental Chronic Duodenal Obstruction, Arch. Surg. **14**:762 (March) 1927.

tion, prolonged stasis, altered motility and an increase in the number of bacteria developed in the duodenum. Under these conditions, examinations were made of the blood of the twelve dogs employed in the earlier investigations. The dogs lived for periods extending from fifty-nine days to one year and forty-five days.

The studies on the blood were conducted in the following manner: At weekly intervals, a full blood count was made. This included hemoglobin determinations, red and white cell counts, differential leukocyte counts and smears for the study of the morphology of the erythrocytes. At the same intervals, the blood was examined chemically for urea nitrogen, carbon dioxide combining power and chlorides. Figures were excluded that were obtained during or after periods of vomiting, or shortly before death. In addition, the gastric contents were aspirated and examined for the presence of free hydrochloric acid in order to determine whether the reverse motility of the duodenum above the obstruction neutralized the free hydrochloric acid by reflux of the duodenal secretions into the stomach.

EXPERIMENTAL RESULTS

Examination of the blood showed that all of the dogs developed a moderate secondary anemia. The maximum fall in red cells varied from 1,000,000 to 2,500,000. The hemoglobin index was always below 1. Frequent remissions occurred when the red cell count was normal. In a control dog kept under the same conditions as the experimental animals for a period of 1 year and 113 days, similar, though less pronounced fluctuations in the erythrocytes were observed. Except for an occasional normoblast and slight anisocytosis, the morphology of the cells remained unchanged. There was no evidence of a blood picture resembling that of pernicious anemia.

The chemical constituents of the blood did not reveal any significant changes during symptom-free periods. Although rather marked fluctuations occurred in the urea nitrogen, comparison of the figures for dogs with duodenal obstruction with those of a series of normal dogs observed over a prolonged period by McKinley [6] showed that the variations in the values were not beyond normal limits. The results of the chemical analysis of the blood at the last examination of the three dogs which were observed for the longest periods are given in table 1.

Examination of the gastric contents always showed that free hydrochloric acid was present. Apparently, the reverse peristalsis which was observed fluoroscopically in the duodenum did not neutralize the gastric acidity by reflux of the duodenal contents. As achylia is characteristic of pernicious anemia, the persistence of free hydrochloric acid

6. McKinley, E. B.: To be published.

in the stomach in the experimental animals may have been of some importance in the results.

PATHOLOGY

The anatomic changes which occur above a chronic obstruction in the intestine in man have been described by numerous observers;[7] however, due to the inability of previous investigators to produce experimental chronic intestinal stenosis, little has appeared in the literature concerning this phase of the subject. Herczel,[8] one of Nothnagel's pupils, studied the effects of incomplete stenosis of the intestine in rabbits. Although none of his animals survived longer than sixteen days, he attempted to interpret the nature of the compensatory muscular hypertrophy which developed in chronic intestinal obstruction. He found that an increase in the thickness of the intestinal wall began as early as the fourth or fifth day after the obstruction was established, and that hypertrophy was pronounced on the ninth day. Lengthening of

TABLE 1.—*Results of Last Chemical Examination of the Blood of Three Dogs Observed for the Longest Periods*

Dog Number	Degree of Dilatation*	Duration of Obstruction at Time of Last Blood Examination	Urea Nitrogen, Mg. per 100 Cc.	Chlorides, Mg. per 100 Cc.	Per Cent of Carbon Dioxide by Volume
8191	++++	1 year, 29 days	22.5	433	48.3
8209	+++	1 year, 23 days	27.0	404	54.8
8418	++++	11 months	17.1	438	53.4

* +++ indicates dilatation approximately twice the diameter of the normal duodenum; ++++ indicates dilation more than twice the diameter of the normal duodenum.

the intestine above the stenosis was associated with the hypertrophy. The other structures of the wall of the bowel did not partake of this change. Herczel concluded that the increased thickness of the intestinal wall above the obstruction was due to a true muscular hypertrophy in the sense of Virchow.

In chronic intestinal obstruction in man, ulceration of the mucosa has been noted frequently in the segment immediately above the obstruction. Patel[9] believed that intestinal hypertrophy occurred only when a lesion of the mucosa was associated with the stenosis, the degree of the hypertrophy depending on the number of excitatory impulses arising from the irritated mucous membrane. In this way he accounted for

7. Nothnagel, H.: Diseases of the Intestines and Peritoneum, Philadelphia, W. B. Saunders Company, 1907, p. 355.

8. Herczel, E.: Experimentelle und histologische Untersuchungen über compensatorische Muskelhypertrophie bei Darmstenosen, Ztschr. f. klin. Med. **11**:321, 1886.

9. Patel, M.: Remarques sur l'état du segment intestinal situé en amont d'une sténose, Lyon méd. **93**:73, 1900.

the hypertrophy of the intestine found in cases of ulceration without stenosis.

The renal lesions that Brown and his associates [4] encountered in chronic duodenal obstruction in man consisted of a "nephrosis characterized either by acute degenerative changes in the tubular epithelium or by a diffuse nephritis."

After the completion of the studies concerning the effects of experimental duodenal obstruction in dogs which were reported previously by us,[5] autopsies were performed and gross and microscopic examinations of the organs were made. This afforded the opportunity to investigate the histologic changes in the duodenum after prolonged stenosis. In addition, in view of the fact that in man changes in the kidneys, liver, gallbladder, joints and hematopoietic organs have been

TABLE 2.—*Duration of the Obstruction, State of Nutrition and Cause of Death of Thirteen Dogs*

Dog Number	Duration of Obstruction	Nutrition	Cause of Death
8190	190 days	Good	Acute ileus
8191	1 year, 44 days	Good	Killed
8209	1 year, 45 days	Good	Killed
8205	113 days	Poor	Acute ileus
8313	73 days	Good	Acute ileus
8314	254 days	Good	Killed
8332	211 days	Poor	Acute ileus
8360	59 days	Good	Killed
8361	151 days	Poor	Acute ileus
8364	161 days	Poor	Acute ileus
8417	84 days	Poor	Acute ileus
8418	1 year, 3 days	Good	Killed
8887	51 days	Poor	Acute ileus

associated with "intestinal stasis and intoxication," a study of these organs in experimental animals was particularly interesting.

METHOD

Immediately after death, the organs were fixed in Zenker's fluid and 10 per cent solution of formaldehyde. Paraffin sections were made and stained with hematoxylin eosin and van Gieson's stain. In addition, frozen sections were made of the liver and kidneys, and were stained with scharlach r for fat.

The twelve dogs studied previously and one additional dog not included in the earlier series constituted the material for pathologic examination. In table 2, the total duration of the obstruction, the state of nutrition and the cause of the death of the dogs is tabulated.

The obstructions existed for periods extending from fifty-one days to one year and forty-five days. Eight dogs died with symptoms of acute ileus. In six of them, the onset of symptoms was characterized by the refusal of food and rapid emaciation, with or without vomiting. In two dogs in which the nutrition remained good (8190 and 8313),

the onset of symptoms was more abrupt, with sudden severe vomiting and prostration. The five remaining dogs were killed during exploratory operations; their nutrition was good. The three female dogs (♀191, 8209 and 8418) became pregnant and had litters during the experiment.

In none of the dogs was the lumen of the duodenum completely occluded at the site of the obstruction at autopsy. The degree of the obstruction did not bear any relationship to the development of symptoms of acute ileus. In the dogs which lived for the longest periods without the occurrence of symptoms, the stenosis was more pronounced than in some of the others that died earlier of ileus. In this connection it was interesting to note the variation in the susceptibility of different dogs to the initial obstruction. After what was considered to be a moderate degree of stenosis had been produced in some dogs, death occurred a few days after the operation, with few objective symptoms; at autopsy it was found that the stenosis was slight or moderate, that the duodenum above the fascial band was not dilated, and that the stomach was normal. On the other hand, other dogs withstood more severe grades of obstruction without difficulty.

In two dogs (8417 and 8418) the lumen of the duodenum immediately above the stenosis was filled by an oval, hard mass which measured 4 by 5 cm., and which consisted of hair and debris matted together. By exploratory operations and fluoroscopic examinations, it was determined that the mass had been present for at least six weeks in the duodenum of dog 8417 before the onset of symptoms of acute ileus. In dog 8418, it had been present for at least eight months without causing symptoms. At fluoroscopy it was noted that the barium flowed around the masses in a thin stream, showing that the obstruction was incomplete.

The usual gross appearance at autopsy of the upper abdominal viscera, after chronic duodenal stenosis, is shown in the accompanying drawing of dog 8418 which had an obstruction for one year and three days (fig. 1). In ten dogs, the stomach was unaltered; in three dogs which developed acute ileus, the stomach was markedly distended. The muscularis was not thickened. The liver, gallbladder and pancreas appeared normal. In some of the animals, the common duct was dilated.

The most significant pathologic changes were found in the duodenum above the stenosis. Dilatation was present in varying degrees; the observations corresponded roughly with the observations which were made at fluoroscopy and exploratory operation.[4] The amount of dilatation in the different dogs varied roughly between one and three times the diameter of the duodenum below the obstruction. The segment of the duodenum above the stenosis was lengthened, owing to the

hypertrophy of the musculature which was always present. The mucosa was normal and in some dogs had a velvety appearance. Ulcerations were found only in the two dogs which harbored masses in the lumen of the intestine; in these dogs, the mucosa was destroyed for a distance of approximately 6 cm. above the obstruction.

There were usually a few thin adhesions at the site of the obstruction, which was further away from the pyloric sphincter than at the original operation owing to the lengthening of the duodenum associated with

Fig. 1 (dog 8418).—Appearance of the duodenum above and below the obstruction which existed for one year and three days. The bulge above the stenosis was due to a large mass, indicated by the dotted circle, which was present in the lumen of the duodenum for at least eight months.

its hypertrophy. The muscularis was not cut through by the band in any case. The lumen of the duodenum at the stenosis was narrowed from one third to two thirds its normal diameter in the fixed specimens. At autopsy, in the fresh specimen, the degree of constriction appeared to be more marked than when the sections were examined. The muscularis was usually thinned out; the mucosa was intact. and

ulcerations were not present. Below the obstruction, the duodenum and the small intestine were normal.

There was no correlation between the degree of the obstruction and the amount of dilatation and hypertrophy of the duodenum. In some dogs, the changes were more marked than in others, although grossly the degree of the obstruction was the same or even less. This corresponded to the variations in stasis and dilatation of the duodenum which were observed fluoroscopically in dogs having approximately the same amount of stenosis.[5] Individual differences in the tonus of the musculature, variations in the ability of the smooth muscle to compensate for the obstruction by hypertrophy and unavoidable differences in the degree of the initial obstruction were probably the factors underlying the variations in the changes found in the duodenum.

TABLE 3.—*Width of Muscularis of Duodenum Above and Below Stenosis*

Dog Number	Width of Inner Circular Layer		Width of Outer Longitudinal Layer		Total Width of Muscularis	
	Below Stenosis, Mm.	Above Stenosis, Mm.	Below Stenosis, Mm.	Above Stenosis, Mm.	Below Stenosis, Mm.	Above Stenosis, Mm.
8190	0.38	1.10	0.16	0.22	0.54	1.32
8191	0.60	0.95	0.18	0.24	0.75	1.19
8209	0.50	1.12	0.19	0.26	0.69	1.38
8295	0.47	0.83	0.21	0.64	0.68	1.47
8313	0.35	1.31	0.14	0.40	0.49	1.71
8314	0.68	0.98	0.27	0.32	0.95	1.30
8332	0.51	1.70	0.21	0.40	0.72	2.10
8360	1.02	1.83	0.29	0.48	1.31	2.34
8361	0.90	2.29	0.26	0.67	1.16	2.93
8334	0.81	1.75	0.29	0.31	1.10	2.09
8417	0.26	0.86	0.08	0.24	0.34	1.10
8418	0.63	1.11	0.19	0.31	0.82	1.42
8887	0.48	1.29	0.15	0.31	0.58	1.60
Average	0.58	1.32	0.20	0.37	0.78	1.69
Ratio	1	2.28	1	1.85	1	2.17

The sections of the duodenum which were selected for comparative histologic study were obtained from the following levels: at or just below the sphincter of Oddi, through the middle of the obstruction and a few centimeters below the latter.

In order to determine the amount of hypertrophy of the muscularis of the duodenum above the obstruction, comparison was made with the width of the muscularis below the obstruction. In addition, the outer longitudinal and the inner circular muscular layers were measured separately for comparison of the relative hypertrophy of each component. The determinations were made in the following manner: The sections of the intestine were enlarged to a known magnification, measurements were made, and after reducing to scale, the results were expressed in terms of millimeters. The measurements were considered to be only relative owing to possible variations in the state of tonus of the intestine at the time of fixation.

The results in thirteen dogs showed that the average total width of the muscularis above the stenosis was 2.17 times greater than the size of the muscularis below the obstruction. A comparison of the separate components revealed that the inner circular layer contributed more than the outer longitudinal layer to the total increase. The relative average increase of the inner layer was 2.28 times, compared with an increment of only 1.85 times of the outer layer. The absolute difference in the cubical increase of the two layers was much greater. In the thirteen

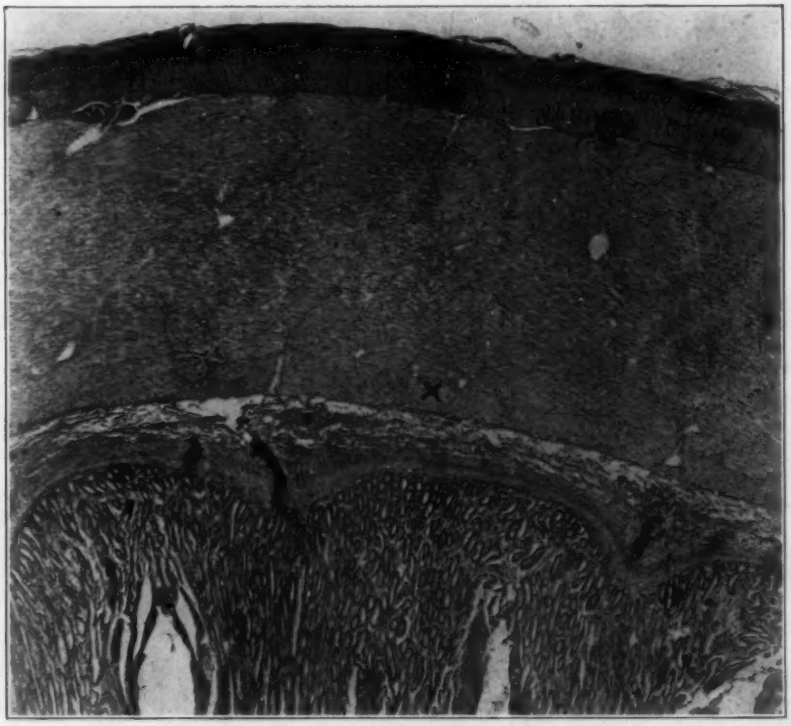

Fig. 2 (dog 8332).—Hypertrophied muscularis of the duodenum above the obstruction which existed for 211 days. Note the specialized layer of muscularis described in the text, indicated by *X*. Compare with figure 3. Reduced from a magnification of × 32.

dogs which were examined, the inner circular layer of the normal duodenum below the obstruction was from two and one-half to three times thicker than the outer longitudinal. Therefore, quantitatively, the bulk of the inner circular layer above the stenosis was increased many times more than that of the outer layer. These changes were more marked in some dogs than in others. The variations are indicated in the chart. The accompanying photomicrographs (figs. 2 to 5) illustrate the appearance of the duodenum above and below the obstruction in

two dogs with well marked hypertrophy. Although the degree of hypertrophy is as marked in dog 8887 as in dog 8332, the duration of the obstruction was only fifty-one days in the former compared with 211 days in the latter. In the sections, the stenosis was less marked in dog 8887 than in dog 8332. Examination of the individual smooth muscle cells of the hypertrophied muscularis showed an increase in length and breadth of the cells with a corresponding increase in the size of the nuclei.

During the course of this study, we observed a muscular layer which has not been described heretofore. This bundle of smooth muscle cells (indicated by *X* in the photomicrographs of dog 8332) was just internal to the inner circular layer. In some dogs it was more marked than in

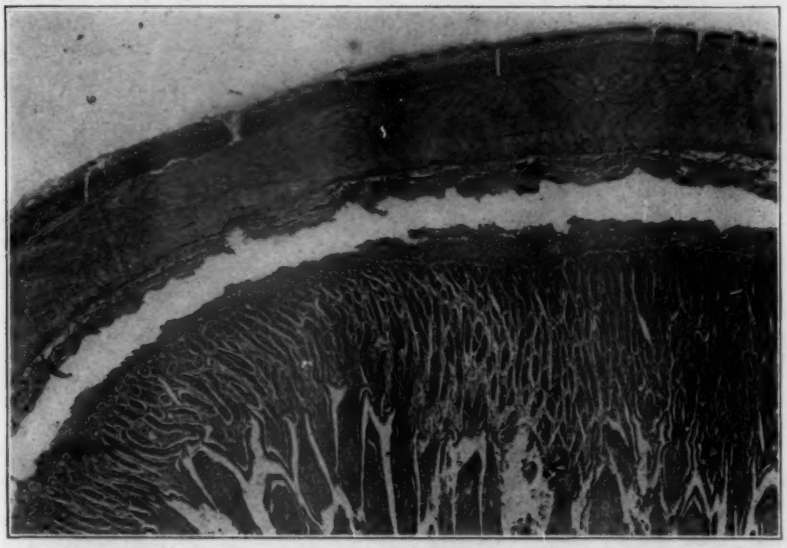

Fig. 3 (dog 8332).—Muscularis of the duodenum below the obstruction. Reduced from a magnification of × 32.

others. In the illustrations, the layer was more definite in dog 8332 than in dog 8887. Its fibers ran parallel to those of the inner circular musculature, but it was distinguished by the fact that the smooth muscle cells were more compactly arranged and the nuclei were more numerous and thinner than those of the circular component. This specialized layer was separated from the adjacent circular one in many instances by a row of cells with deeply stained oval nuclei, and it partook in the generalized hypertrophy (fig. 2). On the other hand, the muscularis mucosae was not increased in width. Changes were not observed in the blood vessels or in the plexuses of Meissner and Auerbach.

Associated with the increased surface area of the lumen of the dilated duodenum above the obstruction, there was hyperplasia of the

mucosa. This marked increase in the amount of mucosa was a striking feature of the obstructed duodenum in all of the dogs; in some, the villi were longer and more tortuous than those below the stenosis, so that the surface area of the individual villi was also greatly increased. Pathologic changes were found in the structure of the mucosa only in the two dogs which harbored large foreign bodies in the duodenum above the obstruction. In these dogs, for a distance of approximately 6 cm. above the fascial band, the greater part of the mucosa and submucosa was destroyed, forming a large ulcer of the Grawitz "decubital" type.

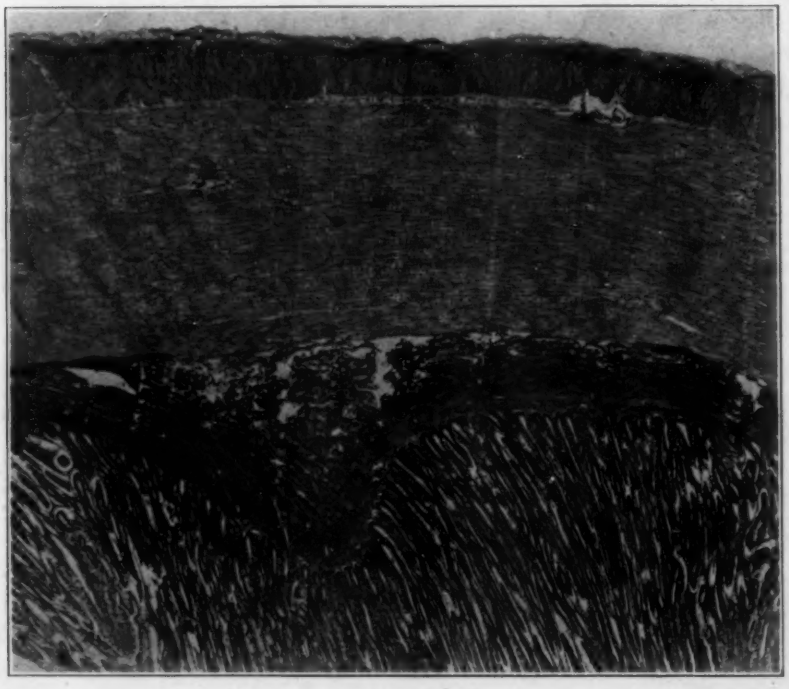

Fig. 4 (dog 8887).—Hypertrophied muscularis of the duodenum above the obstruction which existed for fifty-one days. Compare with figure 5. Reduced from a magnification of × 32.

The base of the ulcer was covered by necrotic epithelium, fibrin and granulation tissue infiltrated with polymorphonuclear leukocytes and round cells; in some places, the inflammatory reaction infiltrated the deeper muscular layers of the duodenum.

At the site of the stenosis the sling of fascia and striated muscle which was employed to produce the obstruction was united to the surface of the duodenum by a layer of connective tissue. The muscularis of the duodenum was of irregular width and showed atrophy of varying degree. The muscularis mucosae and mucosa were unchanged. The

villi were approximately the same size as those below the obstruction. Ulcerations were not found. Below the obstruction, the duodenum was normal.

Lesions were found in the kidneys of ten dogs. The changes varied in degree and extent. There was no correlation between the duration or degree of the obstruction and the severity of the lesions. As a matter of fact, the kidneys of the three dogs with obstructions for the longest periods (8191, 8209, 8418) showed fewer changes than the kidneys of many dogs with obstructions of shorter duration.

The gross appearance of the kidneys varied. In ten dogs, the surface was smooth and of normal color; the capsule was thin and stripped easily. The cortex showed an occasional pin-point abscess or scar. On

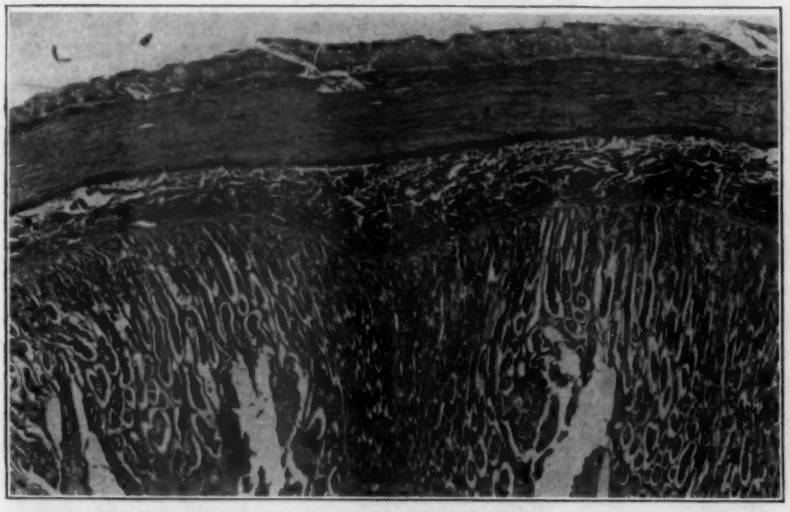

Fig. 5 (dog 8887).—Muscularis of the duodenum below the obstruction. Reduced from a magnification of × 32.

section, the thickness of the cortex was normal; the scars extended for a variable distance into it. In the majority of the dogs, the medullary rays were outlined as thin yellow streaks on the cut surface of the kidney. In three dogs (8313, 8364, 8417), the surface was granular and the capsule was thickened, and in dog 8313 it was adherent; on section, the cortex was irregularly decreased in size, the irregularity being due to scars which extended through the cortex into the medulla; cortical abscesses were also present.

Microscopically, the lesions were found in the cortex and at the corticomedullary junction; in some instances, changes were also present in the medulla. The lesions were usually interstitial and consisted of foci of round cells and connective tissue formation. The accumulation

of round cells varied from occasional small interstitial foci to large abscesses with extensive destruction of the parenchyma; large numbers of polymorphonuclear leukocytes were present in the latter. The proliferation of intertubular connective tissue occurred in the following different forms: as linear scars extending through the cortex into the medulla, as large wedge-shaped areas in the cortex suggestive of healing infarcts and as early lesions consisting of a hyperplasia of young fibroblasts with round cell infiltration. In the areas of fibrosis, the tubules were dilated and distorted. Regeneration of tubular epithelium was observed. In two dogs, calcium deposit was present in a few convoluted tubules in which the epithelium was destroyed. The glomeruli were well preserved as a rule; occasionally, round cell infiltration occurred; in a few dogs, some of the glomeruli were completely obliterated by connective tissue, and in one dog calcification was found. Most of the dogs had accumulations of fat in the epithelium of Henle's loops and the proximal portion of the collecting tubules. The blood vessels did not show any changes.

Microscopic examination of the liver, gallbladder and pancreas did not reveal any pathologic changes. Significant changes were not observed in the bone marrow, spleen or lymph nodes. Evidence was not found of arthritic alterations in the joints. In some of the dogs that died with symptoms of ileus, a terminal pneumonia was found.

COMMENT ON EXPERIMENTAL RESULTS

Although it was well known that hypertrophy of the intestine occurred above a chronic obstruction, experimental studies concerning the part played by the respective layers of the intestines in this reaction had not been published. Although Herczel [8] included some measurements in his work, he did not approach the problem from this point of view. In addition, the duration of the stenosis was too short to draw any conclusions. In the series of dogs which we studied, it was found that the quantitative increase of the inner circular layer of the duodenum was much greater than that of the outer longitudinal layer in the process of hypertrophy. The muscularis mucosae did not partake in the hypertrophy. Hyperplasia of the mucosa occurred, but morphologic changes were not found.

In view of the fact that dogs are subject to naturally acquired chronic nephropathies,[10] the renal lesions which were found in the dogs with chronic duodenal stenosis could not be considered significant. The changes were principally interstitial and may have been due to intercurrent infections or parasites. Evidence of a specific type of lesion

10. MacNider, W. de B.: A Pathological Study of the Acquired Chronic Nephropathy of the Dog, J. M. Research **34**:177 (May) 1916.

was not found in either the tubules or the glomeruli. However, in a series of control animals kept under the same conditions, pathologic changes in the kidneys were far less frequent than in the experimental group of dogs.

Although conditions in the duodenum were made most favorable for the multiplication of bacteria and the elaboration of toxins, systemic effects were not produced. Whether this was due to the ability of the liver to neutralize such toxic substances, remains to be determined. The rôle of the mucosa in this defense mechanism was probably slight, for despite extensive destruction in dogs 8417 and 8418, with excellent opportunities for absorption, the end-results did not differ from those in dogs with an intact mucosa. On the other hand, the dog's liver normally harbors bacteria which have reached it through the portal circulation.[11] In view of the wide margin of reserve which is characteristic of the liver, even if there was increased absorption of toxins and bacteria in the presence of stasis, this natural adaptation to intestinal organisms and their products may have enabled the liver to neutralize the effects of the stenosis.

SUMMARY

Dogs with chronic duodenal stenosis lived for long periods practically symptom free. There was a compensatory hypertrophy of the intestinal musculature above the obstruction, the inner circular layer contributing to the hypertrophy to a greater extent than the outer longitudinal. There was also hyperplasia of the mucosa. Although prolonged stasis and an increase in the bacterial content occurred in the duodenum, significant changes were not observed in other organs.

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STUDIES IN INTESTINAL OBSTRUCTION

I. A COMPARISON OF THE TOXICITY OF NORMAL AND OBSTRUCTED INTESTINAL CONTENT *

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Most clinicians and investigators agree that death in acute intestinal obstruction is due to the absorption of some toxic element from the bowel above the point of obstruction. Much experimental work in the field of intestinal obstruction has been done by American investigators, and it is generally conceded by them that the normal intestinal contents are relatively nontoxic, but that, with the advent of obstruction, a very toxic substance is formed.

Stone, who has done considerable experimental work in this field,[1] states in a recent review [2] that death from intestinal obstruction is due to a toxemia, and that the toxin has its origin in protein decomposition in the obstructed bowel. He says that even after obstruction to the continuity of the bowel has been established, a definite interval of time intervenes before the fluid shows definite evidence of toxicity.[2] From ten to twenty hours usually elapse, according to Stone [2] and Braeye,[3] after the break in the continuity of the intestinal canal has occurred before much fluid accumulates above the obstruction, and thirty-six hours before the fluid shows marked evidence of toxicity. Van Beuren [4] concluded that an interval of forty-eight hours followed obstruction in dogs before the toxic substance accumulated above the point of obstruction. No definite statement appears in the work of many investigators relative to the time interval necessary for the development of this toxic material. But in the light of the intoxication theory which claims most investigators as its adherents, the death of the animal following experi-

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* From the Department of Surgery of the University of Minnesota.
* Presented before the Minnesota Pathological Society, Minneapolis.

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2. Stone, H. B.: The Clinical Application of Experimental Studies in Intestinal Obstruction, Am. J. Surg. **1**:282, 1926.

3. Braeye, Louis: On the Formation of the Toxic Fluid Found in Isolated Duodeno-Jejunal Loops, Bull. Johns Hopkins Hosp. **39**:121, 1926.

4. Van Beuren, F. T.: Enterostomy in Acute Ileus: The Time Element: A Preliminary Report, Am. J. Surg. **1**:284, 1926.

mental intestinal obstruction would indicate that the material above the obstructing mechanism has already become sufficiently potent to extinguish the life of the animal. This time interval varies with the level of the obstruction and the variety of animal. The higher the obstruction, the more virulent is the toxin, and the smaller the animal, the sooner are the effects of the toxin manifested. For high intestinal obstructions in dogs and cats this time interval is usually from forty-eight to seventy-two hours.[5] Occasionally death may occur after twenty-four hours, or it may be delayed ninety-six hours or longer. For rabbits with the same type of obstruction, this interval is usually twenty hours or less. Both rabbits and dogs with obstructions in the colon live considerably longer.

Even though this theory of intoxication as the cause of death in intestinal obstruction is almost generally accepted among experimental workers,[6] a diversity of opinion still exists as to the actual source and nature of the toxin. Whipple and his co-workers[7] believed that the absorbed toxin in intestinal obstruction has its origin in the mucosa of the bowel above the obstruction rather than in the content of the bowel. They believed that the toxic element is in the nature of a heteroproteose. Nesbitt[8] believed that neurin and choline are the toxic bodies that produce the intoxication in obstruction. Gerard[9] stated that the substance is histamine. Murphy and Brooks[10] stated that the action of bacteria is essential for the elaboration of the toxic substance in the obstructed bowel. Dragstedt and his co-workers[11] were also of this

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5. Clairmont, P., and Ranzi, E.: Arch. f. klin. Chir. **73**:696, 1904. Dragstedt, Lester R.; Moorhead, James J., and Burcky, Fred W.: J. Exper. Med. **25**:421 (March) 1917. Draper, J. W.: Intestinal Obstructions, J. A. M. A. **67**:1080 (Oct. 7) 1916. Murphy, F. T., and Brooks, Barney: Intestinal Obstructions, Arch. Int. Med. **15**:392 (March) 1915. Sugito, S.: Mitt. d. med. Fak. d. k. Univ. Kyushu Fuhuoka **9**:229, 1924.

6. Gerard, R. W.: Chemical Studies on Intestinal Intoxication; The Presence and Significance of Histamine in an Obstructed Bowel, J. Biol. Chem. **52**:111 (May) 1922.

7. Whipple, G. B.; Stone, H. B., and Bernheim, B. M.: J. Exper. Med. **17**: 287, and 307, 1913; ibid. **19**:144 and 166, 1914. Whipple, G. H.: Rodenbaugh, F. H., and Kilgore, A. R.: ibid. **23**:123 (Jan.) 1916. Whipple, G. H.; Cooke, J. V., and Stearns, T. J.: ibid. **25**:461 (March) 1917.

8. Nesbitt, Beattie: On the Presence of Cholin and Neurin in the Intestinal Canal During Its Complete Obstruction; A Research on Auto-Intoxication, J. Exper. Med. **4**:1, 1899.

9. Gerard, R. W. (footnote 6): The Lethal Agent in Acute Intestinal Obstruction, J. A. M. A. **79**:1581 (Nov.) 1922.

10. Murphy, F. I., and Brooks, Barney (footnote 5, fourth reference).

11. Cannon, Paul R.; Dragstedt, Lester R., and Dragstedt, Carl A.: Intestinal Obstruction; A Study of the Influence of the Bacterial Flora on the Toxemia of Acute Obstruction, J. Infect. Dis. **27**:139 (Aug.) 1920. Dragstedt, Lester R.; Moorhead, James J., and Burcky, Fred W. (footnote 5, second reference).

opinion. Ellis,[12] Eisberg,[13] Draper [14] (Draper-Maury) and Sweet, Peet and Hendrix [15] believed that the toxic factor responsible for death in intestinal obstruction is allied to the toxemia of pancreatitis.

Comparisons were made in this study between the toxicity of the normal intestinal contents and the contents of the bowel in animals with intestinal obstruction. Comparisons were made of the toxicity of the intestinal contents above and below the point of obstruction. The dog and the rabbit were used in this study.

METHOD

The method employed in collecting the fluid from the bowel was as follows: In the normal animal, the entire content from the stomach to the cecum was pressed into a container by milking the bowel through the gloved fingers. In none of the experiments was the toxicity of the content of the large bowel determined. In the animals with intestinal obstruction, the material from above and below the point of obstruction was collected separately. Obstruction was created in the animals by either severing and turning in the ends of the intestine or by simple ligation of the intestine with gauze from about 20 to 30 cm. beyond the pylorus. This procedure was carried out under aseptic technic and ether anesthesia. The animals were killed when their condition was considered serious, usually from two to three days following the artificial obstruction; in a few instances, the content of the bowel was obtained shortly after the natural death of the animal. In a few dogs closed loop obstruction was performed with and without restoration of the continuity of the intestine. In a few of these, the fluid in the obstructed loop was drained out through a catheter inserted into the bowel. In one instance, the contents of a severed gut obstruction were drained out through a gastrostomy tube.

The intestinal contents obtained in each instance were diluted with an equal amount of physiologic sodium chloride solution. The contents were filtered twice through filter paper under suction. The filtrate was then run through a coarse and a fine Berkefeld filter,[16] and cultures were made to ascertain the sterility of the fluid. The filtration process was started directly after the contents were collected. When injections were made from the same contents on successive days, they were kept in the icebox in the interim. In the instances in which intravenous injections were made in dogs, hemolysis tests were made with the blood of the dog to be certain that the fluid was not hypotonic. The injected material was then clear, often colorless and sterile. The injections were made into the vein on the outer aspect of the dog's leg without anesthesia. Seventeen normal

12. Ellis, J. W.: The Cause of Death in High Intestinal Obstruction, Ann. Surg. **75**:429 (April) 1922.

13. Eisburg, H. B.: Experimental Intestinal Obstruction; A Study in Severed Gut Obstruction and Segmental Obstruction, Ann. Surg. **74**:584, 1921.

14. Draper, J. W. (footnote 5, third reference).

15. Sweet, J. E.; Peet, Max M., and Hendrix, B. M.: High Intestinal Statis, Ann. Surg. **63**:720 (June) 1916.

16. No injections were made with boiled or heated intestinal content nor was any attempt made to determine the toxicity of portions soluble in or precipitated by alcohol.

dogs were injected with intestinal contents prepared in this manner. Of this number, seven were injected with the contents obtained from normal dogs. In five instances, the intestinal contents were used that were collected from above the point of obstruction in four dogs. Material was obtained from below the point of obstruction for four injections from two of these four dogs with severed gut obstruction. In one instance, the intestinal contents were obtained from an obstructed loop drained by a catheter, both ends of the loop having been turned in. A synopsis of the reactions noted are listed in table 1.

TABLE 1.—*Intravenous Injection of the Intestinal Contents Into Normal Dogs*

Dog	Source of Material	Amount Injected, Cc.	Results and Remarks
1	Upper loop of dog with intestinal obstruction	12	Diarrhea; vomiting; salivation; pulse irregular; listless a few minutes after injection; recovery
2	Upper loop of dog with intestinal obstruction	10	Pulse irregular; salivated; frequent defecation; condition satisfactory 4 hours later
3	Upper loop of dog with intestinal obstruction	7	Pulse irregular; listless; not as sick as dog 2
3*	Upper loop of dog with intestinal obstruction	7	Frequent defecation; vomiting and salivation
3†	Closed loops.....	30‡	Not as sick as following second injection; recovery
4	Lower loop of dog with intestinal obstruction	7	Drowsy; pulse irregular; very ill; recovery
5	Normal.....	15	Diarrhea; pulse weak and irregular; labored breathing; comatose; died 3 hours later
6	Upper loop of dog with intestinal obstruction	12	Diarrhea; vomiting; condition satisfactory 3 hours later
7	Normal.....	9	Diarrhea; vomiting; stupor; very ill; died during night
8	Lower loop of dog with intestinal obstruction	15	Slow regular pulse; frequent defecation and vomiting; marked tremors
9	Normal.....	15	Listless; no other effects
10	Normal.....	15	Listless; no other effects
11	Lower loop of dog with intestinal obstruction	15	Mildly ill
12	Lower loop of dog with intestinal obstruction	15	Vomiting; diarrhea; very ill; recovery
13	Normal.....	15	Vomiting; diarrhea; comatose; recovery in 8 hours
14	Normal.....	15	Mildly ill
15	Normal.....	10	Mildly ill
16	Upper loop of dog with intestinal obstruction§	15	Listless; only mildly sick
17	Upper loop of dog with intestinal obstruction§	25	Only mildly sick

* Two days later.
† Three days after second injection.
‡ Drained off through catheter.
§ Obtained through gastrostomy tube.

COMMENT

There were two deaths following the injection of intestinal contents, prepared as described, into seventeen normal dogs. Both deaths occurred following the intravenous injection of normal intestinal contents. Most of the dogs, however, were ill; a few reactions were mild. Listlessness, vomiting, diarrhea, salivation and irregular pulse occurred as a rule. In a few instances, dogs recovered after having exhibited actual stupor and coma.

These reactions noted in so large an animal as the dog are largely qualitative and subject to the criticism that the intravenous injection of any fluid containing protein substances would be expected to elicit reactions. In order to get away from this objection and to obtain better quantiative results, small rats were injected intraperitoneally with intestinal contents prepared in the same manner as that obtained from both dogs and rabbits. The intestinal contents of the dog were used thirty-six times in sixty-six such injections into the peritoneal cavity of the rat, and in thirty instances, the intestinal contents of the rabbit were used. The source of the intestinal content of the dog was the same as that employed in the intravenous injection of the dogs already enumerated. The contents of the rabbit were obtained from the intestine of two normal rabbits and from two rabbits with intestinal obstruction in which the lower ileum had been tied off by gauze two days previously.

TABLE 2.—*Rats Injected Intraperitoneally with the Intestinal Contents of the Dog*

	Number of Rats Injected	Average Amount Injected, Cc.	Sick	Ataxic	Clonic Spasms	Comatose	Died	Recovery
Normal.....	13	5.8	13	3	1	3	1	12
Fluid from above severed gut obstruction	11	5	11	4	1	2	1	10
Fluid from below severed gut obstruction	12	4.8	12	7	3	7	9	3

The rats employed for injection weighed on the average about 75 Gm.; a number weighed as little as 30 and 33 Gm., and a few as much as 200 Gm.

Thirteen rats were injected with the intestinal contents of the normal dog. In eleven instances, the material was obtained from above the point of obstruction in dogs with intestinal obstruction, and in twelve instances the fluid injected was from the loop of bowel below the severed gut obstruction. Thirteen of the thirty-six rats injected died. Nine died following the injection of intestinal contents obtained from the bowel below the point of obstruction. One died following the injection of normal intestinal contents, and another after the injection of contents obtained from above the point of obstruction in a severed gut obstruction.

All the animals injected were ill. Apathy and stupor were the usual symptoms. Ataxia was frequently observed. Clonic spasms were observed in a few instances. Coma was observed when death occurred shortly following the injection. A comatose condition was also observed in a few that recovered. In three instances, death occurred within six minutes from the time of injection; in one instance, three minutes after

injection. In three instances, death occurred from twenty minutes to an hour following injection and in two instances, about two hours following injection. The others died during the night.

Of the thirty rats injected with the intestinal contents of the rabbit, fifteen were injected with material obtained from two normal rabbits, and the other fifteen were injected with material from the two rabbits in which intestinal obstruction had been established by tying off the terminal ileum with gauze two days previously. Five rats injected with the intestinal contents of the normal rabbit and three injected with the contents of the obstructed bowel died. All the animals were ill. Ataxia and clonic spasms and convulsions were frequently noted. The more paralytic and convulsive effect of rabbits' intestinal contents was noted in these injections. One of the rats injected with normal intestinal contents that recovered was rigid with clonic contractions two minutes after injection. Another, also injected with normal intestinal contents,

TABLE 3.—*Rats Injected Intraperitoneally with the Intestinal Contents of the Rabbit*

	Number Injected	Average Amount Injected, Cc.	Sick	Ataxic	Clonic Spasms	Paralytic	Comatose	Died	Recovery
Normal.....	15	6	15	9	6	4	7	5	10
Obstructed...	15	7	15	10	4	2	5	3	12

was ataxic and appeared completely paralyzed three minutes after injection. For a long time the rat lay on his back with flaccid extremities, but it recovered. One rabbit injected with the contents of the obstructed bowel died one minute after injection. Several rats died two and three minutes following injection. The susceptibility of the rat to the injected intestinal fluids did not always vary directly with the amount used nor inversely with the weight of the animal. Instances occurred in which 3 cc. of a given intestinal content killed a fairly large rat when 6 or 7 cc. failed to kill a much smaller one. Postmortem examinations were performed on all the rats that died to rule out puncture of a vessel with consequent hemorrhage as the cause of death. Hemorrhage was not observed in any instance.

Many of the rats that survived injection were reinjected on subsequent days and appeared to have developed an increased tolerance to intestinal contents. We believe that this observation does not have any immunologic significance in treatment for obstruction, but only means that repeated injections of the same protein into the animal increases its tolerance.

The results obtained in these experiments, though at variance with the statements of most investigators in this field, are not without

precedent. Kukula [17] concluded from his experiments on dogs that both normal and obstructed intestinal contents are toxic. Magnus-Alsleben [18] similarly found that the intestinal contents of both the normal dog and the dog with intestinal obstruction was toxic. He observed that the feeding of meat increased the toxicity of normal intestinal contents; the feeding of milk and cheese did not enhance the toxicity. Roger and Garnier [19] stated that normal intestinal contents are even more toxic than the contents from the animal with intestinal obstruction. They observed that the duodenal contents are more toxic than the contents of the lower ileum or colon.[20] Braun and Borrutau [21] also made the observation that in the experimental animal, normal intestinal contents are just as toxic as those from animals with intestinal obstruction.

In this study, we have not attempted to establish comparisons between the toxicity of intestinal contents at different levels of the intestine in the normal animal, nor have we endeavored to ascertain the nature of the toxin in the intestinal contents of the normal animal or the animal with intestinal obstruction. The only deductions that we feel can be made from this study are that the normal intestinal contents of both the dog and the rabbit are toxic on injection when submitted to the same treatment as the contents from obstructed animals. It would also appear that the normal intestinal contents are as toxic as the contents from the obstructed intestine, and that in the dog with intestinal obstruction, the material below the point of obstruction is even more toxic on injection than the fluid obtained from above the obstructing mechanism.

We are aware of the fact that in experiments of this nature there are factors that are not controlled. The dilution of the fluid found in the obstructed and nonobstructed segments is probably the most important. Several investigators have failed to find intestinal contents that have passed through the Berkefeld filter to be toxic. In some rats injected with intestinal fluid obtained from starved animals (both normal dogs and dogs with intestinal obstruction) and prepared as outlined, the symptoms were mild. We also injected twelve rats with the intestinal contents of two dogs in which duodenal obstructions were established

17. Kukula: Untersuchungen üeber autointoxication bei Darmocclusionen, Arch. f. klin. Chir. **63**:773, 1901.

18. Magnus-Elsleben, Ernst: Ueber die giftigkeit des normalen Darminhalts, Beitr. z. chem. Phys. u. Path. **6**:503, 1906.

19. Roger, H., and Garnier, M.: Compt. rend. Soc. de biol. **59**:388, 673 and 677, 1905.

20. Roger, G. H.: Presse méd. **32**:901 (Nov.) 1924. Roger, H., and Josue O.: ibid. **60**:371, 1906. Roger, H., and Garnier, M.: ibid. **60**:666, 1906.

21. Braun, W., and Borrutau, H.: Experimental kritische untersuchungen üeber den Ileus Tod, Deutche Ztschr. f. chir. **96**:544, 1908.

and the dogs given subcutaneous injections of saline solution. Three days later, when, if saline had not been given the animals probably would have been dead, the intestinal contents both from above and below the point of obstruction were collected and prepared as outlined. Twelve rats given these injections, six with the contents from above and six with the contents from below the obstruction, were ill. Two appeared moribund about an hour after injection; one of these was injected with intestinal contents from above, the other with contents from below, the obstruction. The next morning, however, they all seemed active again.

Six rats were then injected with the contents obtained from the intestine of a normal dog diluted with an equal amount of saline. Simple suction filtration alone was employed, the Berkefeld filtration being omitted. All the rats were ill and died a few hours after the injections. Six rats were injected with intestinal contents obtained from a normal rabbit prepared in the same manner. The rats were all ill shortly following the injections and died from its effects.

Such a result, we feel, is to be anticipated because Berkefeld filtration must remove toxic substances.

SUMMARY

The intestinal contents of several normal dogs and rabbits and dogs and rabbits with intestinal obstruction were collected and subjected to Berkefeld filtration. Intravenous injections of the material obtained from the intestine of normal dogs and from dogs with intestinal obstruction were given to normal healthy dogs. Intraperitoneal injections of rats were made from the intestinal contents of both normal dogs and rabbits and dogs and rabbits with intestinal obstruction. Seventeen dogs were injected with the intestinal contents of dogs prepared in this fashion; two died. In both instances, the contents obtained from the intestine of normal dogs was used. Thirty-six intraperitoneal injections of the intestinal contents of the dog were made into rats; eleven deaths resulted. In one instance each, death was due to the injection of the contents of normal and of obstructed loops. The other nine deaths were caused by the injection of intestinal contents obtained from below the point of obstruction into dogs with severed gut obstructions. Thirty rats were injected intraperitoneally with the intestinal contents of the rabbit. Following the injection of fifteen rats with the normal intestinal contents of the rabbit, five died. Of fifteen rats injected with the contents of the obstructed bowel, three died. All the rats that were given injections were ill. The intestinal contents both from above and from below the obstruction in dogs that had been given saline solution subcutaneously was toxic on injection.

CONSTRUCTIONS

1. All intestinal contents are toxic on injection.

2. The intestinal contents of the normal dog and rabbit elicit the same symptoms on injection as do the contents of the obstructed bowel, and are just as toxic.

3. In the dog with intestinal obstruction, the intestinal contents from below the point of obstruction appear even more toxic on injection than the fluid obtained from above the obstructing mechanism.

THIRTY-FOURTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY*

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Concluded from page 168

OPERATIONS ON THE BONES, JOINTS AND TENDONS

Tendon Transplantation for Paralysis of the Opponens Muscle of the Thumb.—Weil [39] used Book's method of tendon transplantation for paralysis of the opponens muscle of the hand and warmly recommended it. The operation consists in transplantation of the extensor of the fifth finger. An incision is made over the dorsum of the fifth metacarpal bone, and the extensor tendons exposed. The extensor minimi digiti is the tendon lying nearest to the ulnar border of the hand. This is freed and divided at the level of the metacarpal head. A second incision is made over the outer side of the first metacarpal bone. The freed tendon is then passed subcutaneously around the wrist and across under the palmar surface to the first metacarpal, to which it is fixed. The thumb is splinted in the opposed position for twelve days, after which exercises and muscle training are started.

Remote Results of Resections of the Elbow.—Sénèque [40] summarized the results of an investigation by Comte on the remote results of resections of the elbow by the method of Ollier for tuberculosis and conditions of ankylosis, which were published as Thèse Lyon, in 1926. These results were:

1. Resections for Tuberculosis: End-results were obtained in 55 patients who had been operated on by Ollier. The time that these

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*This Report of Progress is based on a review of 166 articles selected from 419 titles dealing with orthopedic surgery appearing in medical literature between March 12, 1927, and July 2, 1927. Only those papers that seem to represent progress have been selected for note and comment.

39. Weil, S.: Klin. Wchnschr. **5**:650 (April) 1926.

40. Sénèque: Presse méd. **34**:1351 (Oct. 27) 1926.

patients have been followed varies from five to sixty years, the average being 24.5 years. At the time of operation, 18 of the patients were between 14 and 20 years of age, 24 betwen 20 and 30, and the ages of the rest ranged from 30 to 60. Ollier aimed to restore function, and the results are classified on the functional basis. Complete ankylosis, even though the arm is in good position and useful, is rated as a bad result. The results were found to be as follows: Very good, 26 cases (when the mobility and power exceed two thirds of the normal); good, 15 cases (when one of the two factors mentioned is less than two thirds of the normal but greater than one third of the normal); fairly good, 8 cases (when one of the two factors falls below one third of the normal); bad, 6 cases (more or less complete ankylosis or a flail joint). On the whole, the results were satisfactory in 52 out of 55 cases, or 95 per cent. Perfect results were found in 48 per cent, meaning that the resected side is practically as good as the healthy side, in both power and mobility. Comte pointed out that there is not an absolute relation between the excellence of the anatomic result and of the functional result. In 13 cases, active pulmonary or local tuberculous lesions were present. Two of the patients died six and seven years later.

2. Resections for Ankylosis: Ollier found the results of resection so good in tuberculosis that he applied it as a method for securing mobilization of ankylosed joints. To prevent recurrence of ankylosis, he advised resection of from 5 to 6 cm. of bone and excision of the capsule and periosteal structures about the joint. In 12 resections for arthritis, the results were excellent in 8; good in 2; poor in 1, and bad in 1 (ankylosis recurred, and a second operation was performed later with good results). In 9 resections for fracture or irreducible dislocation, the results were perfect in 3 and bad in 1 (good results were obtained after the second operation). In 5 semiarticular (humeral) resections, the results were excellent in 4 and good in 1. When grouped together, the results were perfect in 76.5 per cent of the cases; good in 19 per cent and poor in 4 per cent. The author compared these figures with the results reported by American surgeons in 126 patients with arthroplasty of the elbow. Good results were obtained in 76 per cent; poor results in 16 per cent, and bad in 6 per cent. Comte concluded that, when compared with arthroplasty of the elbow, the operation of resection appears to be the more successful.

[Ed. Comment.—Comte has made a thorough and painstaking investigation, and the report is deserving of careful study. If a patient with tuberculosis of the elbow can be cured and mobility preserved by resection, this is a better operation than arthrodesis. The operation of

resection was more or less abandoned by American surgeons because of the flail, unstable condition that so frequently resulted. Perhaps this was due to a failure to employ Ollier's careful superiosteal method of dissection. Ollier was a pioneer in operations on the bones and joints and made real contributions to the subject. The report is doubly interesting in that the evidence obtained from a study of Comte's results substantiates his reputation as a master surgeon.]

Transplantation for Paralysis of the Serratus Magnus.—Riedel [41] reported a new method of transplantation of muscle which he employed for paralysis of the serratus magnus muscle with good results. An incision is made extending from the spinal column to the outer edge of the scapula and the latissimus dorsi muscle exposed. The outer half of the muscle is separated from the inner half and its tendinous portion divided. This is then passed under the inner portion of the muscle and pulled through to the lower corner of the inner border of the scapula. The lower part of the muscle is fixed to the sixth, seventh and eighth ribs. In this way, the transplanted muscle is made to act as a muscular ligament for the support of the scapula; it prevents it from sliding off the trunk and permits fair function for the arm.

[ED. COMMENT.—We should want to be convinced that the actions of two different parts of the same muscle can be successfully dissociated before employing this operation.]

Operation on the Hip.—Writing on reconstructive operations on the hip joint, Brackett [42] said that there are three conditions in which opinion differs as to the choice of procedure: 1. In old tuberculous disease, he believed that an operation causing ankylosis offers the only true solution. 2. From the clinical standpoint, he divided patients with osteo-arthritis into three groups: those who have the potentiality of continued function, those who have little, and those with pain and invalidism. In the first group, operation is not necessary. In the second group, the better course seems to be to produce a firm joint which gives stability and relief from pain. In the third group, an operation of stabilization is a matter of convenience to the patient. 3. In old ununited fractures of the hip, the factors to be considered in determining the kind of operation are the degree of atrophy and the size of the remaining portion of the head of the bone, the contour of the head, the appearance of space around the joint, and the presence or absence of osteo-arthritic changes. In these cases, the neck of the femur has practically always disappeared. When the roentgenologic examination gives evidence that the remaining portion of the central fragment is in a condition for performing part of its

41. Riedel, G.: Zentralbl. f. Chir. **54**:578 (March 5) 1927.
42. Brackett, E. G.: Am. J. Surg. **2**:216 (March) 1927.

function, then the best result is obtained by putting it to use, as in the author's operation. When this is not the case, the head should be removed and the neck placed in the acetabulum.

Mathieu and Wilmoth [43] discussed the various operative methods for obtaining ankylosis of the hip in tuberculosis and classified them as intra-articular freshening of the articular surfaces, the extra-articular arthrodesis by rigid bone grafts and the para-articular method of transplanting the greater trochanter. They favored either the latter method, which is essentially the Hibbs operation, or a method with osteoperiosteal grafts, in which a flap of bone is turned down on the capsule from the ilium, and the space between this and the bared trochanter filled in with osteoperiosteal shavings from the opposite tibia.

An Operation for Stabilizing Paralytic Hips.—In a preliminary report, Dickson [44] described an operation for stabilizing paralytic hips which he used for five years. The procedure is designed to replace the paralyzed glutei muscles by the tensor fasciae femoris. He transplanted the origin of the tensor fasciae femoris with a piece of its bony attachment to a groove in the crest of the ilium near the posterior superior spine. The muscle is passed through a tunnel beneath the gluteus medius. The indications for the operation, according to Dickson, are: (1) flexion contracture of the hip; (2) relaxed subluxating hip; (3) holding of a luxated hip in the acetabulum after reduction. The author reported having performed the operation in forty cases, details of which are to be presented in a later paper.

Operation for the Relief of Paralysis of the Gluteus Maximus Muscle.—Ober [45] devised an interesting and apparently successful operation for paralysis of the gluteus maximus muscle. An incision is made from the level of the first lumbar vertebra, 1 inch (2.5 cm.) lateral to the spinous processes, downward opposite the posterior inferior spine of the sacrum, exposing the gliding fascia over the muscles in this region. The fascia is incised longitudinally near the spinous processes and is cleared from the muscle. The outer half of the erector spinae, with its aponeurosis, is separated from the inner half and from the muscles lateral to it, the incisions being carried down to the lower end of the incision in the skin. The muscle thus split off is freed from the body and crest of the ilium, from the sacrum, and from the transverse processes in such a way that a free flap of muscle, about 5 inches (12.7 cm.) long, 1 inch wide, and ¾ inch (1.8 cm.) thick is obtained.

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43. Mathieu, P., and Wilmoth: J. de chir. **28**:130 (Aug.) 1926.

44. Dickson, Frank D.: J. Bone & Joint Surg. **9**:1 (Jan.) 1927.

45. Ober, Frank R.: Operation for Relief of Paralysis of Gluteus Maximus Muscle, J. A. M. A. **88**:1063 (April 2) 1927.

An incision is next made over the lateral aspect of the thigh from the tip of the trochanter to 1 inch above the level of the patella, exposing the tensor fasciae latae and the fascia lata. The tensor is freed as in Legg's operation, but the incisions in the fascia are carried down the leg as far as the incision in the skin extends in order to form a long flap of fascia 1 inch wide. A hole is drilled through the femur just below the neck at the level of the gluteus maximus tendon, and the long flap of fascia is drawn through the hole from before, backward. At that point the edges of the fascia are sutured to the gluteus maximus tendon. The free end of the fascial flap is then drawn up under the gluteal fascia to the lower angle of the first incision, care being taken to keep the gliding surface of the fascia next to the iliac bone. The free end of the erector spinae muscle is laid on top of the fascial flap which had been scarified, so that they overlap 2 or 3 inches, and then the edges of the fascial flap and the aponeurosis of the erector spinae muscle are sutured under moderate tension; thus, extension of the tendon is made from the erector spinae muscle to the femur. The leg is put up in a long plaster spica.

Since July, 1926, the author has not bored the transverse hole below the trochanter for the purpose of inserting the fascia, especially in those cases in which power is absent in the tensor fasciae latae; his technic consisted of dissecting the strip of fascia up to the insertion of the gluteus maximus tendon, and then following out the technic already described in passing the tendon up over the buttocks. Indications for the operation are: (1) paralysis of the gluteus maximus muscle; (2) paralytic dislocation of the hip, and (3) flexion deformity of the hip when associated with paralysis of the gluteus maximus.

Late Repair of Fractures of the Patella and of Rupture of the Ligamentum Patellae and Quadriceps Tendon.—Gallie and LeMesurier [46] described in detail operations for the repair of the conditions already mentioned. They pointed out that while these injuries frequently heal solidly and appear firm within a few weeks, in a certain number the scar may begin to weaken and stretch so that after a year all power of extension has gone. Repair is accomplished in the case of injury of the patellar tendon by taking heavy transplants from the Achilles tendon and passing them through the patella to the tibial tubercle. In the case of ununited fractures of the patella, fascial strips are used when the bone fragments can be approximated, but transplants from the Achilles tendon are used when a gap remains. In all patients with a ruptured quadriceps tendon, whether the ruptures have occurred early or late, Gallie and LeMesurier advocate repair by the use of fascial suture.

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46. Gallie, W. E., and LeMesurier, A. B.: J. Bone & Joint Surg. **9**:47 (Jan.) 1927.

[Ed. Comment.—We wish to record our agreement with the recommendations of the authors, but we go farther in that we advise the method employing the fascial suture in the repair of acutely fractured patellae as well as in the old ununited fractures. This method has been employed at the Massachusetts General Hospital for from three to four years, and it has been possible to allow motion and use from the start without the necessity of any splinting.]

Synovectomy in Chronic Arthritis of the Knee Joint.—In cases of chronic infectious arthritis of the knee, when improvement has not been obtained from focal and other methods of treatment, Payr [47] recommended excision of the synovia. He performed this operation in seven cases with satisfactory results. The synovia was found markedly thickened, sometimes to the size of a finger, and contained granulomatous masses, areas of necrosis, and foci of bacteria. Payr felt that there is little danger of ankylosis or of secondary infection.

Operative Lengthening of the Tibia and Fibula.—Abbott [48] reviewed the work on operative lengthening of the tibia and fibula and described his apparatus for lengthening both bones of the leg. It is modeled somewhat after Putti's apparatus and consists of two steel transfixion pins held in adjustable rods fixed to a Thomas splint in such a way that absolute control of the fragments is obtained. His operation consists of the following steps: (1) lengthening of the Achilles tendon; (2) osteotomy of the fibula; (3) insertion of pins; (4) osteotomy of the tibia, and (5) application of stabilizers and foot-piece. The lengthening is accomplished by a gradual adjustment of the apparatus so that from one-sixteenth to one-eighth inch a day is gained. The stretching is maintained for about three weeks, or until about 2 inches (5 cm.) is gained. Of six patients treated, results in five were considered successful by the author, from 1¼ to 1⅞ inches (3.1 to 4.6 cm.) having been gained. He pointed out that rigid asepsis must be maintained and that the surgeon who has not the time to make daily mechanical adjustments should not try the method.

[Ed. Comment.—The author is employing the operation for lengthening bone not only in cases in which the full length of the limb has previously existed and has been lost, as in the case of malunited fractures, but in cases in which the full length of the limb has never existed, such as in infantile paralysis and old epiphysitis. His work demonstrated that a gain of 2 inches or more can be obtained in such cases without harm. The procedure is fraught with danger, however, from first to last and demands the closest attention from the surgeon.

47. Payr, E.: München. med. Wchnschr. **74**:1437, 1927.

48. Abbott, LeRoy C.: J. Bone & Joint Surg. **9**:128 (Jan.) 1927.

It should not be undertaken until the technic has been acquired by actual observation of Abbott's methods.]

A Plastic Operation on the Foot.—Miller [49] described a new operation on the foot which he applied to patients with adolescent relaxed feet which do not respond to the usual conservative measures. The operation consists of a plastic transplant of the insertion of the calcaneoscaphoid ligament to the first cuneiform and metatarsal, together with arthrodesis of the scaphoid-cuneiform and cuneiform-first metatarsal joints, and occasionally resection of the neck of the astragalus. He also lengthened the Achilles tendon. Plaster is worn for six weeks, after which an adducting shoe with the Thomas heel is worn. The author reported satisfactory results in sixteen cases.

Flatfoot and Its Treatment.—Von Dittrich [50] reported the results of Mueller's operation in twenty-five patients with severe flatfoot. Mueller's operation consists in drilling a hole through the tarsal scaphoid and pulling the tendon of the anterior tibial muscle, or a part of it, through that hole. The results have been satisfactory. In the author's opinion, more extensive operations ought not to be employed unless repeated manipulation has demonstrated the impossibility of correcting the position of the foot and hence the necessity of an operation on the bony structures.

Talipes Cavus.—Rugh,[51] who felt that contracture of the plantar fascia is the primary cause of pes cavus and the cause of relapse and of continued progression of the deformity, advised operative excision of the plantar fascia and its replacement by the transplantation of fat tissue that does not contract. After observation of the results of this procedure over a period of four years, he was convinced that it is possible to prevent contraction of the scar by this method and that it does not lead to sagging of the plantar arch.

[ED. COMMENT.—This would appear to be a radical method for accomplishing the same result that is obtained by stripping the plantar ligaments from the os calcis.]

Results of Operation for Hallux Valgus.—Mau and Lauber [52] examined a group of patients who had been operated on for hallux valgus at the surgical clinic in Kiel, to determine the end-results. In fifteen cases the operation had consisted of the simple excision of the exostosis, and the results were generally unsatisfactory. In twenty-nine cases Hueter's operation, which consists of resection of the head of the

49. Miller, Oscar Lee: J. Bone & Joint Surg. **9**:84 (Jan.) 1927.
50. Von Dittrich, K.: Deutsche Ztschr. f. Chir. **196**:194, 1926.
51. Rugh, J. T.: Am. J. Surg. **2**:307 (April) 1927.
52. Mau, C., and Lauber, H. J.: Deutsche Ztschr. f. Chir. **197**:361, 1926.

first metatarsal bone, had been performed, and from a study of the results the authors felt that this procedure involves the risk of causing further spreading of the anterior arch or of producing stiffness in the joint from resulting bony proliferation. The best results were seen in thirty cases in which Ludloff's operation—oblique osteotomy of the first metatarsal bone—had been performed. Even here the results were not entirely satisfactory, and the authors recommended a combination of Ludloff's operation with transplantation of the abductor hallucis. They considered that proper after-treatment is of great importance.

Pick [53] studied a group of 28 patients who were operated on in the city hospital of Dortmund from twelve to fifteen years previously. Of these, 14 were well pleased with the results; 11 were barely satisfied, and 3 were dissatisfied. The best results were seen either after Ludloff's operation (as already described) or after Hohmann's operation, which consisted of a wedge-shaped excision posterior to the head of the first metatarsal bone with transplantation of the abductor hallucis muscle.

Cleveland [54] reported the results of about 200 operations for hallux valgus in 108 patients. Five different types of operation had been employed: (1) simple excision of the exostosis; (2) wedge-shaped osteotomy of the metatarsal with excision of the exostosis (Ludloff's operation); (3) resection of the proximal end of the phalanx with excision of the exostosis (Keller's operation); (4) resection of the metatarsal head with excision of the exostosis (Hueter's operation), and (5) excision of the exostosis with division of the lateral ligaments and of the adductor muscles and realinement of the phalanx on the metatarsal by means of a flap formed from the medial ligament (Silver's operation). The author found that in this series resection of the metatarsal head with removal of the exostosis had given the most satisfactory results. The most important cause of failure of any operation is the presence of chronic arthritis in the joint, and in these cases the results are poor in proportion to the severity of the arthritis. Other causes are a severely impaired anterior arch, inadequate excision of the metatarsal or phalangeal exostoses, and failure to remove loose fragments or bone sand. When the sesamoid bones show hypertrophic changes, they should be removed. Of greatest importance is the selection of an operative procedure that fits the individual case.

[Ed. Note.—It is interesting to have these studies of end-results from such widely separated communities. They agree in condemning simple removal of the exostosis. The German reports praise the results

53. Pick, H.: Arch. f. Orthopaedie, 1926, vol. 35.

54. Cleveland, Mather: Hallux Valgus, Arch. Surg. **14**:1125 (June) 1927.

of Ludloff's operation, while the American report favors Hueter's procedure. Studies of end-results are often disappointing in that they fail to provide any basis for generalizations. If we were to draw any conclusion from these reports, it would probably be to the effect that the method of operation is of less importance than the skill with which it is performed and the care that is employed in the after-treatment. It is also important to suit the operation to the individual, especially with reference to age.]

MISCELLANEOUS

Sprain of Cervical Spine Causing Thrombosis of Anterior Spinal Artery.—Discussing the mechanism of vertebral sprains in the cervical region, Grinker and Guy [55] pointed out that as a result of trauma, transitory derangement of the normal relationship of one vertebra to another may occur, the vertebrae quickly returning to normal alinement. This is accompanied by a stretching and laceration of the spinal ligaments. Only when the vertebrae do not return to their original relationship is the condition recognized as a luxation. Greuber reported, with necropsy, an example of vertebral sprain due to excessive muscular exertion which produced thrombosis of a portion of the anterior spinal artery in the cervical region and which was followed by softening of the cord in the region supplied by the artery. The authors concluded that temporary vertebral luxations may occur and give rise to lesions of the cord without any evidence by roentgenologic examination of vertebral displacement.

[ED. COMMENT.—This case is important as an example of the actual pathologic process that may underlie some of the cases of obscure injury of the back.]

Rupture of the Supraspinatus Tendon.—Although admitting that he has never performed an early operation on or seen a patient with rupture of the supraspinatus tendon soon after injury, Codman [56] advocated early operations for exploration when the lesion is suspected. He based this advice on his own experience of finding at operation that the tendon had been ruptured in many of the old traumatic shoulders. He estimated that this lesion makes up a third of injuries of the shoulders. The signs of the lesion are inability to abduct the arm actively, a slight jog in the motion of passive abduction, and tenderness and soft crepitus beneath the acromion anteriorly. Codman approached the shoulder in the majority of his cases through a split deltoid incision, but extended this into the "saber cut" incision when necessary.

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55. Grinker, Roy R., and Guy, Chester C.: Sprain of Cervical Spine Causing Thrombosis of Anterior Spinal Artery, J. A. M. A. **88**:1140 (April 9) 1927.

56. Codman, Ernest A.: Boston M. & S. J. **196**:381 (March 10) 1927.

The Rôle of the Capsule in Contractures of the Joints.—Silver [57] discussed the arrangement of joint capsules and pointed out that from a surgical standpoint one must consider the capsule as including not only the supporting synovial membrane but the ligamentous extensions and bands attached about the joints. He further emphasized that the capsule is largely on the flexor surfaces of the joints. He divided capsular contractures into four types: simple postural, traumatic, inflammatory and trophic. He advocated subperiosteal separation of the capsule by the subcutaneous method for relief of contractures of the fingers, the metacarpophalangeal joints, the wrist, the elbow, the ankle and by the open method at the knee. The incision for the knee is made laterally just in front of the biceps tendon and the capsule is stripped, care being taken to free the capsule in the intercondyloid notch.

Tendovaginitis Stenosans of the Wrist.—Winterstein [58] gave a detailed description of tendovaginitis stenosans at the styloid process of the radius, stating that the condition is not rare and should be readily recognized. The main symptom is pain at the radial styloid and is due to the narrowing of the tendon sheath containing the tendons of the long and short abductors of the thumb. The pain is increased by movements of the wrist and thumb, and there is local tenderness on pressure. The condition may heal within a few weeks as a result of conservative treatment, but frequently remains obstinate. In such cases prompt relief can be obtained by operation, the sheath being split or a small strip excised.

Treatment of Ischemic Contraction of Hand.—Johansson [59] reported three advanced cases of ischemic paralysis in which complete functional recovery was obtained by operative treatment. This consisted of the freeing of all the flexor muscles and lengthening of all the flexor tendons. The results were compiled three and one-half, four and four and one-half years after operation.

Osteitis Deformans Framboesica.—Matsunga [60] reported twenty-four patients with frambesia who showed skeletal changes typical of osteitis deformans.

Actinomycosis of Bone.—Krogius [61] reported a case of actinomycosis of the tibia without any primary focus elsewhere. The symptoms simulated osteomyelitis, and the diagnosis was not made until three

57. Silver, David: J. Bone & Joint Surg. **9**:96 (Jan.) 1927.
58. Winterstein, O.: München. med. Wchnschr. **74**:13 (Jan. 7) 1927.
59. Johansson, S.: Acta chir. Scandinav. **61**:188 (Dec. 22) 1926.
60. Matsunga, T.: Acta Dermatologica **2**:76 (Jan.) 1927.
61. Krogius, A.: Finska läk.-ällsk. handl. **69**:1 (Jan.) 1927

months after the onset. The patient was treated with roentgen-ray irradiations and potassium iodide. The case terminated fatally one year later.

That actinomycosis is not as rare as is generally believed is shown by the report of four cases of vertebral actinomycosis by Simpson and McIntosh.[62] These patients were encountered at the Pathological Laboratory of the University of Michigan, two within the past year. All came to autopsy with a clinical diagnosis of tuberculosis of the spine. According to the authors the characteristic lesion in actinomycosis of the spine is cortical erosion of the vertebrae with vertebral phlegmon. Central destruction of the body of the vertebra, with ultimate collapse and deformity such as is found in tuberculosis, does not occur. The roentgenograms should readily distinguish the two conditions. Radical surgical excision of all involved tissue offers the best hope of cure.

The Snapping Knee.—In an extensive paper describing in all fifty-five cases with twenty-two operations, Kuettner and Liebig[63] discussed the problem of the snapping knee. They distinguished three groups of cases: (1) cases not showing a known injury, in which the cause is either congenital or in connection with subluxation of the tibia forward or with genu recurvatum; (2) cases following distinct trauma, in which the most common cause is displacement or tearing of the lateral semilunar cartilage, less frequently of the medial cartilage, or in which rupture of one or both crucial ligaments may be present; (3) cases due to exostoses.

Clinical experience and experiments on the cadaver led Ostrowski[64] to the conclusion that the snapping knee is commonly due to displacement of the lateral meniscus or to the rupture of the anterior crucial ligament or both. The cause is generally of traumatic nature, though the snapping may not begin until some time after the trauma. The only treatment is by operation with excision of the cartilage or repair of the torn crucial ligament.

Roentgenography of Joints with Iodized Oil.—Sievers[65] employed iodipin for the roentgenologic examination of joints, and his article is illustrated by excellent roentgenograms, especially of the joints of the hips. In congenital dislocation of the hips, many details, which remain uncertain in the ordinary films, can be shown by this method. The width of the isthmus, the size of the acetabulum and of the cartilaginous head and, especially, the results of reduction can be determined accurately.

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62. Simpson, Walter M., and McIntosh, C. Alexander: Actinomycosis of Vertebrae, Arch. Surg. **14**:1166 (June) 1927.

63. Kuettner, H., and Liebig, F.: Ergebn. d. Chir. u. Orthop. **19**:439, 1926.

64. Ostrowski, S.: Beitr. z. klin. Chir. **136**:591, 1926.

65. Sievers: Fortschr. a. d. Geb. d. Röntgenstrahlen, 1926, vol. 35.

In cases of osteochondritis, the defects in the articular surfaces are clearly shown. Sievers felt that there may be some therapeutic benefit in certain cases from the slow absorption of the oily substance; on the other hand, there is the disadvantage that additional roentgenograms may be indistinct.

[ED. COMMENT.—Sievers' method would appear to be of value for purposes of research but not for routine clinical investigation.]

Some Anomalies and Deformities of the Vertebrae.—Hanson [66] made a study of roentgenograms of vertebrae of different ages, beginning with the fetal period. From the latter part of fetal life up to the second year, the body of the vertebra is divided into three plates, the upper and lower ones being dense, with a lighter one between. At the anterior border of the latter, a conelike cavity is seen, with its base forward. Anatomically, this corresponds to a channel in the bone containing bone marrow and a vein, and is not a nutrient foramen, as formerly stated. The cavity disappears at the end of the second year in all except the fifth, sixth and seventh dorsal and first lumbar vertebrae. In these vertebrae the cavities usually disappear at the fourteenth year, but occasionally persist even up to the twenty-fifth year. Hanson believed that the frequency of tuberculous lesions in these vertebrae depends largely on the persistence of the cavities, as they contain many blood vessels. Injection of the nutritive arteries shows that one artery supplies two adjoining vertebrae, and this may account for the frequent involvement of the adjacent vertebrae by a tuberculous process.

FRACTURES

Leukocytosis Accompanying Fractures.—Walton [67] made a study of the occurrence of leukocytosis in 260 patients with fracture and drew the following conclusions: Traumatic injury of bone gives rise to a moderate leukocytosis, which is chiefly polymorphonuclear in type. The increase in white cells reaches a maximum within twenty-four hours following the injury and remains approximately at this level until immobilization of the fragments is secured. Reduction and immobilization are followed by a rapid fall in the white cells, with a return to normal at the end of the fifth or sixth day. Old, debilitated persons usually develop less leukocytic reaction than do children or young adults. The increase in the number of white cells appears to be largely the result of injury to the bone and periosteum but varies also in proportion to the amount of injury of the soft tissues and displacement of fragments.

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66. Hanson, R.: Acta chir. Scandinav. **60**:309, 1926.

67. Walton, Ralph W.: Leukocytosis Accompanying Fractures, J. A. M. A. **88**:1138 (April 9) 1927.

Statistical Study of Fractures Received During the War.—Continuing his studies of the casualties in battle among the American forces in the World War, Walker [68] stated that there were 147,651 gunshot injuries, of which 25,272, or 17.12 per cent, were fractures. Of these men, 2,751, or 11 per cent, died. The total loss of time due to gunshot wounds was 14,544,536 days, and 35.24 per cent of this time, or 5,125,220 days, was due to fractures. The average time lost for each fracture of the femur received during battle, from which the patient recovered, was 326.9 days; for each compound fracture not received in battle, 238.8 days, and for each simple fracture not received in battle, 184.4 days. Of the men who had fractures of the femur, only 158, or 3 per cent, were returned to duty. Five per cent were discharged from the hospital in less than 300 days after being injured; 24 per cent, in less than 400 days, and 71 per cent required treatment in the hospital for more than 400 days. The number of wounded men discharged from the army on account of disability was 25,187, and of these 11,740, or 47 per cent, were discharged because of fractures. In all there were 32,331 individual fractures received in battle, giving an excess of fractures over patients of 7,059.

[ED. COMMENT.—With recollections still fresh of the fractures received in the battles in France, of what extensive and horrible injuries they usually represented, and of the trying conditions under which the patients had to be treated, we are gratified to learn that the mortality rate did not exceed 11 per cent. We consider this a tribute to the treatment which these patients received.]

Longitudinal Fracture of the Neck of the Radius.—Phillips and Galland [69] called attention to a hitherto undescribed fracture of the neck of the radius consisting of a longitudinal split extending from the epiphyseal line at the neck of the radius close to its external margin, distally to the cortex of the bone. They collected reports of five such cases. The fracture is not ordinarily accompanied by any displacement. The origin of the fracture is difficult to explain on anatomic grounds. It probably results from indirect violence and apparently is produced by a fall with the elbow flexed and the forearm pronated. It is most likely an avulsion fracture from the pull of the supinator brevis muscle, a part of which is inserted in the area to which the injury is confined. The treatment is simple support in the Jones position, and a rapid return to full function usually results.

Fractures of the Transverse Processes of the Spine.—From a study of ten patients with fracture of the transverse processes of the lumbar

68. Walker, J. S.: U. S. Vet. Bur. M. Bull., April, 1927, p. 293.

69. Phillips, Herman B., and Galland, Walter I.: Longitudinal Fracture of the Neck of the Radius, J. A. M. A. **88**:1318 (April) 1927.

vertebrae, Kennedy [70] concluded that such injuries are not rare, that the cause is usually direct violence and that the fractures are frequently multiple. He believed that the fracture is negligible and that the disability is largely due to the associated sprain or contusion. Definite bony union is obtained in some of the cases. Disability lasting longer than six months is out of the ordinary, and the majority of the patients should be back at work within two months, practically without complaints referable to the injury.

RESEARCH

Physiologic Study of the Blood Supply of the Diaphysis.—Johnson [71] carried out a well conceived series of experiments to show the relative importance of the different sources of blood supply of the diaphysis. He studied the rate and character of repair in drill hole defects of the cortex in the bones of dogs, under the following conditions: (1) with the nutrient artery intact and all other sources of blood supply cut off; (2) with the metaphyseal vessels alone intact and (3) with the periosteal blood supply alone intact. Specimens have been obtained from the different animals at different periods and the reparative process followed. From these experiments, Johnson concluded that the nutrient vessels maintain nutrition throughout the medulla and the inner half of the cortex. Repair is active when the blood supply of the bone is derived from these vessels. The metaphyseal vessels supply the medulla and the inner half of the cortex. With the blood supply from other sources cut off and these vessels alone intact, repair is active but not so active as with the nutrient vessels. The periosteal vessels do not supply more than the outer half of the cortex. With blood supplied by these vessels alone, repair is greatly delayed and its character altered. Thus the nutrient vessels are the most important, the metaphyseal vessels next, and the periosteal blood supply the least important.

Regeneration of the Synovia.—Wolcott [72] performed experimental synovectomy on the joints of dogs. The animals had been killed at periods ranging from 1 to 108 days and the joints studied. Gross examination, roentgenologic study of the joints injected with sodium iodide, and microscopic investigation showed the formation of a new synovial layer and the restoration of a practically normal joint pouch within 108 days. The newly formed membrane is produced by metaplasia of the connective tissue, and when the membrane has been formed this process is inhibited.

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70. Kennedy, Robert H.: Ann. Surg. **85**:519 (April) 1927.
71. Johnson, Robert W.: J. Bone & Joint Surg. **9**:153 (Jan.) 1927.
72. Wolcott, W. Eugene: J. Bone & Joint Surg. **9**:67 (Jan.) 1927.

[ED. COMMENT.—Wolcott's results are in agreement with the experimental observations of Key, to which attention has been called in a previous "Report of Progress in Orthopedic Surgery."]

Seeliger [73] performed experiments on rabbits to determine the fate of blood in the joints. He was unable to show the presence of any special ferment that would prevent clotting of blood inside the articulation. He concluded that whether or not clotting takes place after injury with intra-articular bleeding depends entirely on the function which the joint is permitted. If the joint is placed at rest, clotting occurs. Later, the cellular elements are eliminated and the serum absorbed. If, on the other hand, motion is permitted, clotting does not occur, and the action is similar to defibrination of blood outside the body, which is produced by stirring or beating it in a glass container.

Experimental Radium Arthritis and Articular Neoplasm.—Experimenting on rabbits by the introduction of radium into the joints, Fisher [74] produced changes that were characteristic of arthritis. In one rabbit an intra-articular periosteal sarcoma developed.

Roentgenologic Examination of the Shoulder.—King and Holmes [75] studied 450 roentgenograms of the shoulder in an effort to find an answer to the question of why the roentgen-ray examination gives negative results in such a large number of shoulder cases. Of the films examined, 66.6 per cent were interpreted as giving negative results. The authors concluded that this high percentage of negative results may be materially reduced by a more careful physical examination to exclude cases in which the pain is referred to the shoulder from lesions in other situations. Further, they advised making one underexposed film as well as a normal one with the shoulder externally rotated in order to demonstrate insertion fractures of the supraspinatus and subacromial bursitis.

Influence of Surrounding Tissue on the Fate of Transplanted Bone.—Wereschtschinski [76] performed a series of experiments on animals to determine the influence of surrounding tissues on transplanted bone. For transplantation, he employed autoplastic bone pegs and took pains to have them fresh, living, and covered with periosteum. The recipient tissues were changed as little as possible. Cultures of bone tissue in the test tube did not show new growth of bone. Implantation of bone into the soft tissues was followed by only slight formation of new bone, and

73. Seeliger, P.: Klin. Wchnschr. **5**:1616, 1926.

74. Fisher, A. G. T.: Brit. M. J. **1**:319 (Feb. 19) 1927.

75. King, J. M., Jr., and Holmes, G. W.: Am. J. Roentgenol. **17**:214 (Feb.) 1927.

76. Wereschtschinski, A. O.: Westnik chir. i. prog., 1926, p. 84.

the process of absorption soon increased and surpassed it. When the bone was implanted in the marrow cavity, it occasionally healed in place, but in the end it was absorbed and the marrow space reestablished. Only when the bone was implanted on bone did firm consolidation and union of the transplant with the recipient tissue take place. An actual metaplasia of connective tissue into real bone has been seen by the author following transplantation of fine splinters of bone into the soft tissues. Ten days after the transplantation, the splinters were surrounded by a thick layer of osteoid tissue.

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